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THE

AMERICAN YEAR-BOOK

OF

MEDICINE AND SURGERY

BEING

A Yearly Digest of Scientific Progress and Authoritative
Opinion in all Branches of Medicine and Surgery,
drawn from Journals, Monographs, and Text-
Books of the Leading American and Foreign
Authors and Investigators

COLLECTED AND ARRANGED

WITH CRITICAL EDITORIAL COMMENTS

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UNDER THE GENERAL EDITORIAL CHARGE OF

GEORGE M. GOULD, M.D.

MEDICINE

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PREFACE.

THE death of Dr. Charles H. Burnett and the resignations of Dr. Ingals and Dr. Ohls have allowed the union of the departments of Otology and Laryngology under the editorship of Dr. D. Braden Kyle and Dr. George Fetterolf. Subscribers interested in these specialties will be glad to have them brought in this way into their proper logical and clinical combination, and especially under the care of one so exceptionally fitted for the work as is Dr. Kyle. The continued success of these Year-Book volumes, despite so many old and new rivals, is a source of gratification to the editors.

GEORGE M. GOULD.

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GENERAL MEDICINE.

By ALFRED STENGEL, M.D., and D. L. EDSALL, M.D.,
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INFECTIOUS DISEASES.

GENERAL CONSIDERATIONS REGARDING INFECTIOUS DISEASES.

Firket[1] has made a study of the diseases common in Europe as they are seen in the tropics. He notes that **smallpox** is the **plague of almost all the hot countries,** and that in India there is scarcely a child that has not had the disease, which, it is believed by the natives, constitutes a necessary means of purification of the body. Extremely grave epidemics are common, and, at times, kill off a large percentage of the inhabitants of certain regions. The peculiarity of smallpox in hot countries is the **frequent recurrence of the disease;** recurrences are, however, usually mild. The author considers that insects are of importance in spreading the infection in the tropics, largely because they are so extremely numerous. Vaccination is being used more frequently in the tropics, but it is important to have vaccine stations and to avoid carrying the vaccine from Europe, as it is likely to lose its efficacy in transportation. One difficulty met in doing this, however, is that the Hindoos, for instance, will not allow cattle to be used for this purpose.

Influenza has no especial peculiarities in the tropics. **Acute rheumatism** is not so common as in Europe; Europeans in the tropics more frequently have rheumatism, and also cardiac involvement, than do the natives. **Pneumococcic infection** shows some peculiarities; many observers believe that the disease in natives often shows severe general symptoms, with very slight local involvement. This question, however, has not been properly studied.

Typhoid fever has been thought to be very rare in hot countr[illegible] which malaria was common, although malaria with intestinal lesion[illegible] not infrequently described. Now it seems that typhoid is becoming [illegible] common, as malaria is being, to some extent, stamped out; typhoid [illegible] is, at any rate, everywhere present. It occurs more frequently in E[illegible]peans than in natives, but this is probably due to the fact that the la[illegible] have acquired some immunity through having been infected in childh[illegible] In Algiers the intestinal lesions seem to be less [illegible], or even abs[illegible] with great frequency; and the disease runs under the picture of a typhoid septicemia, like pneumonia in negroes.

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[1] Bull. de l'acad. royale med. de Belgique[illegible]

Menzer[1] contributes a discussion of the **etiology of acute infectious diseases,** particularly in their **relation to tonsillar infection.** His earlier investigations concerning infection in rheumatism through the tonsils were somewhat disappointing; but he describes later results which showed, in a case of rheumatic sore throat associated with fresh endocarditis, diplococci in the peritonsillar tissue. In two other cases he also found diplococci and streptococci; and he insists upon the importance of this method of infection, and considers that the lymphatic ring about the throat and fauces is intended for the purpose of shielding the organism from the entrance of microorganisms through the mouth and nose. This, however, is frequently insufficient. One may, then, get a mere sore throat with muscular pains, or may get various other conditions, particularly rheumatism; the conditions, under these circumstances, not being so much the result of a specific infection as of the reaction of the individual. At times, however, specific microorganisms gain entrance through the tonsils; and the author believes that this is a comparatively common occurrence. He thinks, for instance, that genuine pneumonia probably often arises in this way, and does not consider it demonstrated that this disease usually arises through aspiration.

M. Beniasch[2] contributes a study of the **therapeutic value of antipyresis** based upon a quantitative determination of the agglutinating power of the blood in typhoid fever. His purpose was chiefly to determine whether this method would aid in showing the truth or incorrectness of the present teaching that fever in infectious conditions has a useful curative influence upon the organism. He decides that there is absolutely **no evidence that this teaching is correct,** and that his results rather speak directly to the contrary. The agglutinating power of the blood does not show any corresponding rise with the rise of the temperature, or fall with the fall of the temperature, either when antipyretic treatment was used or when it was not used. He discusses the animal experiments that have been done upon the question, and criticizes all those that have apparently demonstrated the curative value of fever. He considers it important to determine the influence of antipyresis upon those elements which have been determined to have a close relation to the protection from or the destruction of infectious principles; viz., the antitoxins, bactericidal substances, agglutinating substances, etc.

TYPHOID FEVER.

Etiology.—H. E. L. Canney[3] discusses the theory of **air-borne typhoid fever in armies,** particularly considering the reports concerning the disease at several stations in India, where the infection was supposed to have occurred at least partly through the air. He criticizes the reports because they overlook various important means of infection through water, such as washing vegetables in infected water, the opportunity afforded for the men to have drunk infected water, etc.; and ends with the conclusion that the evidence does not establish the occurrence of air-borne infection, the weight of evidence being in favor of its being water-borne. He refers to his own experience in Egypt, which gives a very interesting demonstration of the entire disappearance of typhoid fever when a body of persons is entirely protected from water-borne infection, although the opportunities for air-borne infection still exist. He believes that infection through flies, dust, etc., may occur; but that it is certainly extremely rare.

---

[1] Berl. klin. Woch., No. 1, 1902.

[2] Zeitschr. f. klin. Med., Bd. XLV, Hefte 1 und 2

[3] Brit. Med. Jour., Aug. 24, 1901.

W. E. Harriman[1] reports an **epidemic of typhoid fever** at the Iowa State Agricultural College. The college is situated in excellent surroundings, the buildings are large and well constructed, and the water-supply planning and sewage-disposal are good. The epidemic was apparently traced to **a portion of the milk-supply,** a case of the disease having occurred in the family of the milkman. At the college 42 cases were treated, 2 dying, and 23 were treated at home, of whom 3 died. The large proportion of the cases treated at the college were severe. Intestinal hemorrhage occurred in 7 of the 42 cases. Phlebitis occurred in 5, one patient having it in both legs. Neuritis was present in 4. In 3 patients the temperature passed 106° F. and in 1 case it was over 107° F. for 3 hours. Intestinal perforation occurred once, and acute mania once, the latter in a rather mild attack. The author notes that in 3 cases copious hemorrhage occurred immediately after enemas, and that in one case there were repeated small hemorrhages after each attempt to use an enema. Through this experience he has been led to fear their use.

B. K. Rachford[2] reports an epidemic of 35 cases of typhoid fever at a summer-resort in Michigan. It was **traced to a manhole receiving sewage** and kitchen-water, which communicated with two tanks, in one of which vegetables and food were placed for cooling, and in the other of which water for drinking purposes was collected. On pumping out the latter tank, water was seen to ooze in from the neighboring manhole. When the leak was corrected, the cases ceased to develop.

R. H. Quill[3] discusses typhoid fever as it **occurred in a camp** in Ceylon in 1900. He believes that all the water-supply had been efficiently guarded, and that the infection in the camp must have been air-borne from infected latrines or infected dust, or carried by flies.

E. F. Brush[4] contributes an interesting study of an epidemic of typhoid fever in an **extremely isolated settlement** in the backwoods of Maine. The drinking-water was determined to be the source of the infection.

Symptomatology.—H. Lüthje[5] has carried on some interesting investigations concerning **metabolism in 2 cases of typhoid fever.** He studied the cases during the course of the disease, during the early period of convalescence, and in a period quite a long time after the convalescence from the acute disease. His chief purpose was to determine the nature of the nitrogen-retention which is well known to occur in convalescence from acute disease. He reaches the definite decision that the use of abundant food which contains a large amount of albumin causes a very **decided and prolonged nitrogen-retention,** even in normal persons; and the figures were such as to indicate that the retained nitrogen could not be *in toto* due to muscle formation, or even have been retained in the tissues united with water, in the proportion in which nitrogen and water are normally found together in the tissues. This is directly contrary to the usual physiologic teaching, that the nitrogen-retention goes to form tissue—chiefly muscular tissue. Lüthje believes that a large portion of the nitrogen thus retained cannot form tissue. He also investigated the influence of different forms of nitrogenous food, using meat and casein as examples of two different varieties, his chief reason for this being the fact that it had been suggested by himself and others that casein is present in milk—the natural infants' food—because it is more easily assimilated than meat albumin, even though it is somewhat more difficult of digestion. His results spoke directly against this view, much more of the albumin contained in meat being retained than of an equal amount of protein in the form of casein. He also had an opportunity to observe the influence of menstruation in one case, and found, as others have, that it causes a decided nitrogen-retention. The figures obtained in one of the cases during convalescence, when, because of an attack of severe tonsillitis, there was a 9 days' period of extreme subnutrition or almost starvation, were very striking. During the period of hunger the patient lost 438 grams of albumin, equivalent to over 2 kg. of muscle. The same amount was regained in the first four days of convalescence from the tonsillitis.

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[1] Jour. Am. Med. Assoc., Feb. 22, 1902.

[2] Medicine, Dec., 1901.

[3] Brit. Med. Jour., Feb. 15, 1902.

[4] N. Y. Med. Jour., April 5, 1902.

[5] Zeitschr. f. klin. Med., Bd. IV, Hefte 1 und 2.

A. Kuhn and A. Suckstorff,[1] in a general **contribution to the statistics** and to the pathology and therapy of typhoid fever, describe an epidemic which occurred in 1900 in Rostock. The epidemic itself was of considerable severity. The cases were decidedly more prevalent in September, October, and November than in other months. The mortality was 13.4 %, which is higher than it had been there for some years. There occurred about 1 case to every 965 inhabitants. The authors discuss 90 cases, 53 of them being men and 37 women. They note that there were several instances of infection in the hospital—one nurse, one orderly, and one patient. The nurse and the orderly were both engaged in the care of typhoid cases. There were 6 cases of hemorrhage, of which 3 were fatal. Relapses occurred in 18.8 %. The Widal reaction was positive in 95 %; hypoleukocytosis was present in 90 %; splenic tumor in 86.6 %; the diazo reaction in 75 % to 85 %; roseola in 69 %. The authors place the value of different symptoms in the early diagnosis of typhoid fever as follows: (1) Hypoleukocytosis; (2) Widal reaction; (3) roseola; (4) diazo reaction; (5) enlargement of the spleen. They note the frequency of the occurrence of a wild delirium in their cases, in spite of the use of tub-baths, 21 % exhibiting maniacal delirium. They note one case of paralysis of the deltoid muscles—an unusual situation. They had no satisfactory results with the specific antityphoid treatment. In the treatment of hemorrhage they recommend the use of hydrastis canadensis. They have used gelatin, but have not seen any evidence of a good effect in cases that were at all severe. They warmly recommend the use of urotropin in typhoid for disinfection of the urine.

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[1] Deut. Arch. f. klin. Med., Bd. LXXI, Hefte 4 und 5.

H. Allyn,[1] in reporting the **statistics of 184 cases** of typhoid fever occurring at the Philadelphia Hospital, states that the mortality was 16.84 %, the high percentage being attributed to the fact that the hospital admits a very wretched class of persons. Death was due to asthenia in 14 cases, to intercurrent affections in 8, to hemorrhage in 5, and to perforation in 3.

H. D. Rolleston,[2] in discussing **enteric fever in the hospital at Pretoria,** notes that there were a number of cases in persons who had previously had typhoid fever elsewhere, and thinks that these repeated attacks may be due to infection with a different strain of typhoid bacillus. Antityphoidal inoculations did not absolutely protect against enteric fever, but the disease did not seem to occur within 6 months after the inoculation, and none of those who were inoculated died. [The figures are, however, altogether too insignificant to permit of the conclusion drawn, that inoculation protects against a fatal termination of the disease. The figures are very unconvincing.] Relapse occurred in 21 %. Diarrhea was exceptional. The pulse-rate during convalescence was often extremely rapid. This was attributed to the physical overstrain which had usually preceded the attacks. This symptom was favorably influenced by tincture of belladonna. Hemorrhage occurred in 8.7 %, and 76 % of the cases in which it occurred were fatal. A scorbutic tendency was often noted. Perforation occurred in but 1 of the 244 cases; phlebitis was common; and edema and increased firmness of the leg, without pain or tenderness and without evidence of cardiac weakness, were common during convalescence, and may have been due to **thrombosis of the deep veins.** The frequency of the phlebitis was thought to have been possibly due to the fatigue consequent upon "trekking." Two fatal cases of **gangrene of the leg** were observed; bronchitis was rare; pneumonia occurred in 4 cases; urticaria was very common. One case of localized myositis is described. There were 3 cases of orchitis, 1 of jaundice, and 1 of solitary abscess of the liver with recovery; the latter occurred in a man who had never had dysentery.

R. Proust[3] discusses **recurrences of typhoid fever in the same individual.** These usually occur in epidemics or as a result of an evident direct source of contagion. They are much more frequently observed in men than in women. The symptomatology does not differ from that of primary attacks, except that the second attack is likely to be milder than the first. Abortive, ambulatory, and extremely mild cases constitute a large proportion of second attacks. The diagnosis is often very difficult, because of the patient's having previously had an attack. The prognosis is commonly good. High fever is uncommon, and nervous symptoms are, as a rule, absent. Convalescence is generally rapid.

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[1] Phila. Med. Jour., Dec. 7, 1901.

[2] Brit. Med. Jour., Oct. 5, 1901.

[3] Gaz. des Hôp., No. 137, 1901.

E. Bendix and A. Bickel[1] make the very interesting statement that in a case of typhoid fever complicated by severe symptoms they found on each palatoglossal arch and on the epiglottis an elevated, plaque-like area, while the surrounding tissues were unaffected. They introduced a platinum needle into one of these plaques, after carefully sterilizing the surface, and obtained cultures of a bacillus that was identical with the typhoid bacillus, and that gave the serum-reaction with the blood of another patient with typhoid fever. The plaques ulcerated slightly, but soon healed. This seems to be the first satisfactory *intra vitam* demonstration of **typhoid bacilli in typhoid throat lesions.**

Opie and Bassett[2] report the case of a child of 10 who was taken sick with diarrhea and pain in the abdomen, and then ran through the course of a typical typhoid fever, with the exception of the disappearance of the intestinal symptoms. The child died on the twenty-sixth day of the disease, after the occurrence of hemorrhages in the skin and mucous membranes and severe epistaxis. Typhoid bacilli were isolated from the blood and from various organs. There were **no intestinal lesions found;** the follicles were scarcely swollen, but were slightly pigmented. The abdominal lymph-glands and the spleen were, however, decidedly enlarged. Such cases are probably explained through the previous existence of intestinal lesions which have already healed. At any rate, there is no positive proof of invasion of the organism with typhoid bacilli through the intestine without lesion of that organ.

Complications.—Of interest in connection with perforation is the report of A. Oppenheim[3] on **disappearance of the liver dulness** without visible meteorism. He has determined to his satisfaction that one can inflate cadavers and cause the liver dulness to disappear, without evident marked distention. He also distended the intestines of 3 normal women, and found that the liver dulness practically completely disappeared. He attributes this to a rotation of the liver upon its axis, and not to its being covered with intestine. One result of this rotation of the liver, since it cannot move bodily backward, is to cause compression of the lungs or to push them aside—a point of importance in the differential diagnosis of a number of conditions of the chest and abdomen. In one experiment, in which air was pumped in, under pressure, through a tube passed into the stomach, the author found a marked distention of the abdomen, which was practically due entirely to distention of the small intestine; this caused no reduction in the liver dulness, and practically no change in the position of the liver.

C. Bacaloglu[4] discusses the **conditions of the heart in typhoid fever,** partly from an experimental and partly from a clinical standpoint. Intraperitoneal injections of typhoid bacilli in animals cause only slight myocardial lesions, but intracardial infection causes constant lesions of the pericardium and myocardium, and often of the endocardium also. The cardiac complications of the disease seem to be **about as common in children as in adults, although less severe.** The signs which indicate myocarditis are disturbance of the ratio between temperature and pulse, gallop rhythm, impure first heart-sounds, vomiting, unexplainable dyspnea, and slow pulse. Arrhythmia is not infrequent in the course of typhoid fever, but is not, of itself, a bad prognostic sign. It not infrequently indicates the beginning of convalescence. Cardiac infarct is rare in typhoid fever. Thrombosis is somewhat more common. Sudden death occasionally occurs in the third or fourth week, frequently preceded by syncope; and is at times, although by no means always, due to myocarditis. The prognosis in typhoid fever depends much more upon the condition of the pulse and heart than upon the temperature. Typhoid pericarditis occasionally occurs alone, but is more frequently combined with endocarditis. Whether it is due to the Eberth bacillus or not, is uncertain. It is often difficult to diagnose. A fatal case is mentioned. When it occurs, endocarditis usually appears in the second week, and commonly involves the mitral or tricuspid valve. It is practically always —perhaps always—caused by other microorganisms than the Eberth bacillus. In treating cases of typhoid fever, if the signs of myocarditis appear and become marked, **cold baths should be stopped;** if the cardiac signs are mild, however, and the general symptoms are severe, the baths may be continued, together with the use of heart tonics; if endocarditis or pericarditis occurs, the baths should be stopped. Wet or dry cups the author considers valuable in endocarditis or pericarditis, but not in myocarditis. An ice-bag over the precordia is useful. Fractional doses of digitalis he considers of value, as is also caffein. If collapse occurs, baths should be absolutely stopped.

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[1] Deut. med. Woch., June 5, 1902.

[2] Bull. Johns Hopkins Hosp., July, 1901.

[3] Deut. med. Woch., July 3, 1902.

[4] Gaz. des Hôp., 1901, No. 78.

P. Michel[1] discusses **typhoid pleurisy.** He makes the following division of the cases: (1) A secondary pleurisy following a lung affection, such as pneumonia, bronchitis, or infarct; (2) an actual typhoid infection of the pleura through the lungs; (3) isolated pleurisy which is not due to the typhoid bacillus, but is the result of infection with the tubercle bacillus, streptococcus, or other organism, in a subject of typhoid fever. Typhoid pleurisy **almost always causes exudation;** the latter is, however, usually slight, and is likely to be localized in the posterior portion of the lung, or to be interlobular or diaphragmatic. It **tends to spontaneous cure,** and does not, as a rule, cause any notable symptoms—not even pain; it is, consequently, apt to be overlooked. Pleural typhus, in which the picture of pleurisy occurs in the course of the disease, is rare; pleurisy occurs much more frequently in convalescence. Owing to the fact that the exudate is not likely to be large, and that the symptoms are slight, typhoid pleurisy is **often overlooked,** and frequently can be diagnosed only by exploratory puncture. In the metatyphoid form, however, the symptoms of onset are likely to be much more severe. The disease lasts a variable time, usually as long as 6 weeks. Typhoid pleurisy does not cause any marked change in the prognosis of the disease.

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[1] Gaz. des Hôp., 1901, No. 87.

K. Hühn and M. Joanovic[1] report a case of **suppuration of echinococcus cyst of the liver** produced by typhoid bacilli 4 months after the onset of the typhoid fever. The signs of the suppurative condition began with sudden icterus and pain in the gastric region, and the appearance of a tumor, which was discovered by the patient. The tumor increased and the skin became reddened over it. There was fluctuation over the mass. The skin became edematous. An aspirating needle was introduced and pus was obtained. The incision was then carried out, and the operator discovered a suppuration in a cavity containing multiple small cysts. The echinococcus cyst was looked upon as being a *locus minoris resistentiæ,* in which the typhoid bacilli had collected and produced suppuration. The clinical diagnosis had been liver abscess, the echinococcus cyst not having been suspected. Cultures and Widal reaction showed both that the patient had actually had typhoid fever and that the bacilli were actually typhoid bacilli.

T. McCrae[2] reports 3 cases of **mastitis** in typhoid fever; and, after a study of the literature, determines that it is a very rare complication, and that it usually occurs late in life, affects both sexes, is not associated with the functionating gland, and involves both breasts in about half the cases. Suppuration occurs in about half the cases, and typhoid bacilli may be found in the pus. The condition is of no special moment in prognosis.

Davis, Patterson, and Hewlett[3] report the case of a woman who, during convalescence from typhoid fever, developed **mastitis, which went on to suppuration.** The abscess was opened, and the pus showed a pure culture of typhoid bacilli.

C. J. Lewis,[4] in discussing the bacteriologic examination of the urine in zymotic diseases, states that **two forms of typhoidal bacilluria** must be distinguished. In one, the condition begins at the height of the disease, is accompanied by albuminuria,—often pyuria,—and not infrequently hematuria occurs in severe cases. The second form, which may succeed the first, begins in the later stages of the disease, and is often protracted for a long time without producing severe symptoms. It may occur in severe or mild cases, and it may not be accompanied by albuminuria, even cystitis not being always present. In 45 cases of typhoid fever the author found but one instance of typhoid bacilluria. In 16 cases of scarlet fever he found streptococci in 7 instances, these organisms being of two forms. In 17 cases of diphtheria he found twice a bacillus morphologically similar to the Klebs-Löffler bacillus, but showing a different staining reaction. In 3 the streptococcus was present; and in some of the cases, other organisms.

Bürsing[5] reports a case that is of decided **interest in relation to public health.** A soldier who had typhoid fever in China in October was, in the following April, found at 4 examinations to have numerous typhoid bacilli still present in his urine, without any disturbance of his health. These bacilli vanished after the use of urotropin.

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[1] Liecnicki viestnik, No. 2, 1902; Centralbl. f. innere Med., No. 13, 1902.

[2] Bull. Johns Hopkins Hosp., Jan., 1902.

[3] Am. Jour. Med. Sci., Dec., 1901.

[4] Edinburgh Med. Jour., Dec., 1901.

[5] Deut. med. Woch., June 19, 1902.

L. Levi and A. Lemierre[1] report a case of **cystitis due to the typhoid bacillus,** the signs of cystitis appearing on the nineteenth day of the disease. The bacteriologic investigation showed the presence of typhoid bacilli, which persisted for over 3 months after their appearance, but disappeared after the use of urotropin. After studying the literature the authors state that typhoid cystitis usually appears toward the end of the disease, often after febrile albuminuria has been present; and that, as a rule, it is associated only with the presence of pus and typhoid bacilli, although irritative symptoms and even hematuria may appear. The urine is always acid. The complication develops rapidly, and, as a rule, is readily overcome by treatment; although sometimes it is very persistent. The typhoid bacilli may be associated with the colon bacillus. The infection is of the most marked importance in its relation to the prophylaxis of typhoid fever.

L. Dickinson[2] describes a case of typhoid fever in a woman of 29, in which there was a good deal of **hemorrhage from the vagina** on admission, on the twelfth day. In the fifth week she bled profusely from the gums and passed blood from the bowel, and there were numerous ecchymoses and, later, hematuria. She **died from hemorrhage,** and necropsy showed widely scattered hemorrhages in nearly all the organs. There was no alcoholism in the case.

C. B. Longenecker and J. Akerman[3] report a case of typhoid fever with a **general hemorrhagic tendency,** treated with calcium chlorid, gelatin, and adrenalin chlorid. The patient recovered.

W. Ewart[4] reports a case of typhoid fever with a general hemorrhagic tendency. It proved fatal.

Vergnau[5] discusses **typhoid fever in tuberculosis.** The presence of the latter disease often has no influence upon the course of the former, but it makes the diagnosis doubtful in many cases. Absence of spots, less marked stupor, marked muscle pains, hyperesthesia of the skin, and a lower and less regular temperature, speak for general tuberculous infection. The immediate prognosis in typhoid fever is not much influenced by the existence of tuberculosis, unless the latter is severe. When phthisis occurs in a convalescent from typhoid fever, the diagnosis may again be very doubtful. Tuberculosis is likely to appear after a brief fever-free interval, and is likely to be mistaken for a relapse of the typhoid. The occurrence of tuberculosis in convalescents is suggested by persistent frequency of the pulse, by a temperature curve suggesting tuberculosis; and by sweats, loss of appetite, emaciation, and the development of pulmonary signs. The prognosis in these cases is bad, as a rule, the course usually being rapid. The author considers it important to recognize the fact that it is **readily possible for tuberculous subjects to acquire typhoid fever in a hospital; and typhoid subjects may readily acquire tuberculosis.** It is, therefore, **extremely important to isolate all cases of each of these diseases from cases of the other;** and in caring for typhoid patients, every one who either is evidently tuberculous, or is suspected of being so, should be excluded.

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[1] Gaz. Hebd. de Méd. et de Chir., No. 98, 1901.

[2] Lancet, July 6, 1901.

[3] Am. Med., Jan. 25, 1902.

[4] Lancet, July 6, 1901.

[5] Gaz. Hebd. de Méd. et de Chir., No. 73, 1901.

E. Mackey[1] reports a case of **tuberculous meningitis** in which the Widal reaction in a dilution of 1:20 was repeatedly obtained. [There was no bacteriologic investigation for coincident typhoid infection, and the previous history did not state whether the patient had had typhoid fever previously or not.]

P. K. Pel[2] reports a case in which there was an attack of acute polyarthritis that was followed by a condition diagnosed as typhoid fever, during which there occurred a relapse of the arthritis. He believes that it was a case of that rare coincidence—**typhoid fever and acute rheumatism.** The Widal reaction was negative throughout.

W. G. Robertson and C. C. Biedert[3] report a fatal case of **Ludwig's angina complicating typhoid fever.** Tracheotomy was done. The course was extremely rapid, the patient dying 10 hours after the onset of the complications; the immediate cause of death was obscure.

T. Diller[4] reports a case of **meningomyelitis** occurring during convalescence from typhoid fever.

J. H. Burch[5] states that he has repeatedly met cases that **closely resembled typhoid fever,** that showed the diazo reaction, and that frequently showed in the urine an organism resembling the Eberth bacillus; but whose blood-serum gave no reaction with the Eberth bacillus, while it did react with the bacillus found in the urine. He describes a case in which the bacillus was determined to be B. coli communis.

Diagnosis.—L. Brieger[6] reports that he has **isolated a specifically acting substance from typhoid bacilli.** Pure ammonium sulfate is made slightly alkaline by adding ammonium bicarbonate in extremely weak solution, and a little ammonium carbonate. The mixture is constantly shaken. The author then pours upon the surface a 3 or 4 days' old agar culture of virulent typhoid bacilli and shakes the mass, when the bacilli form balls. They are placed in the dark for several days; and the bacteria are then filtered off, dried with filter-paper, suspended in an extremely weak soda solution, and shaken for half an hour. The mixture is now centrifugated, and the supernatant fluid is filtered twice through a pukall filter. The fluid contains some substance which, when injected into the animal organism, causes the development of a large amount of agglutinin; but it does not itself agglutinate typhoid bacilli and does not interfere with their growth.

A. Schültze[7] reports his study of the **effect of this substance upon the animal organism,** and finds that it does cause the development of a very marked agglutinating power in the blood of animals that have been injected with it, the agglutination occurring in dilutions as great as 1:1000. As stated, the substance does not itself produce agglutination. It also causes the development of a precipitin. The most important result of this study is the demonstration that this substance apparently has absolutely no influence upon the immunity of the animal. This leads Schültze to decide that there is positively no relation between typhoid agglutination and typhoid immune bodies.

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[1] Lancet, Jan. 25, 1902.
[2] Berl. klin. Woch., April 21, 1902.
[3] Amer. Med., Dec 28, 1901.
[4] Amer. Med., June 7, 1902.
[5] N. Y. Med. Jour., May 31, 1902.
[6] Deut. med. Woch., July 3, 1902.
[7] Ibid.

W. Savage,[1] in discussing **pseudo-clumping in cultures of the typhoid bacillus,** states that a series of cultures from one source will often show itself to have much less tendency to agglutinate than a series of cultures from another source; but, further, the medium used is often at fault. Spontaneous agglutination occurs only in bouillon cultures; and, indeed, the specific agglutination occurs better in bouillon than in agar cultures.

F. Lobiesen[2] discusses the **value of the Widal reaction** in typhoid fever. He **reports 350 cases** which were, from a clinical standpoint, certainly typhoid fever. Of these, 328 gave a positive reaction at 1:50, either at the first or at subsequent examinations; 17 showed a reaction with a dilution of 1:10 or 1:25, but were always negative at 1:50; 2 gave no reaction at any time, even with a dilution of 1:5; 2 other cases gave a positive reaction only at 1:5. Of the 350 cases, 37 ended in death. In 31 of these the diagnosis could be confirmed by necropsy. As to the importance of the reaction in diagnosis, he directs attention to the fact that **289 of the patients exhibited the reaction within the first 40 hours after their admission** to the hospital; in 215 of these instances the reaction was at once positive at a dilution of 1:50. He has examined 151 patients who had not typhoid fever, but who were admitted to the hospital with a diagnosis of typhoid fever, or suspected typhoid fever. The cases proved to belong to a variety of classes: tuberculosis, pneumonia, influenza, pyemia, enteritis, and various other conditions. Of these, 4 gave a positive reaction at 1:25; 2 gave a positive reaction at 1:10, and 123 were negative at 1:10. Of the 4 that gave a reaction at 1:25, 3 died, and showed, respectively, acute miliary tuberculosis, retroperitoneal abscess, and calculous pyelitis with tuberculous meningitis. The fourth patient had pleurisy, and was discharged cured. He has also **examined 61 healthy persons** who had never had typhoid fever: 1 gave a positive reaction at 1:25; 8 gave a positive reaction at 1:10; 52 were negative at 1:10. It is therefore evident that persons may give a positive serum-reaction, either when in health or when suffering from other diseases than typhoid fever, at a dilution of 1:25. He believes that the diagnosis of typhoid fever can be made positively when reaction occurs with a dilution as great as 1:50. It is evident that the absence of the reaction does not always exclude typhoid fever. As a rule, the reaction appeared at the end of the first or beginning of the second week. In 290 patients he noticed that 138 gave the reaction during the first week, 111 in the second week, 30 during the third week, 11 during the fourth week. **Relapses frequently increased the agglutinating power** of the blood, in some instances very decidedly. He also **tested a series of 20 persons who had previously had typhoid fever** from 1 to 23 years before the examination. Of these, 9 had been examined during their illness, which had occurred from 1 to 2 years previously. They had reacted at that time in dilutions of from 1:200 up to 1:700. All these persons reacted at the later period mentioned, one of them in a dilution of 1:50; 5 in dilutions of 1:25; 3 in a dilution of 1:10. All the others also gave a reaction, in only one instance, however, in a dilution greater than 1:10. He considers the reaction of the greatest value in diagnosis, but subject to the criticisms mentioned.

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[1] Jour. Path. and Bact., Nov., 1901.
[2] Zeit. f. klin. Med., Bd. XLIII, Hefte 1 und 2.

Mariotti Bianchi[1] has carried out a series of investigations of the Widal reaction. In 51 cases, in which a dilution of 1:20 was used, 48 were positive. In the cases which were negative the failure of the reaction was attributed to the severity of the disease. In 175 cases other than typhoid fever in which a dilution of 1:20 was used, the author found a positive result in 23 instances. Of these patients, 10 had had typhoid fever earlier, but 13 of them had no history of that disease. In **167 cases of typhoid** a dilution of 1:40 was used; 164 reacted positively, the three negative cases being proved by autopsy to be typhoid fever. Bianchi found that the reaction vanished entirely in one case within 15 days after the disappearance of the fever, and was always very much reduced within 3 months. He considers that if the reaction at 1:40 **appears within 5 minutes, it is of decidedly good prognostic importance.** With a dilution of 1:40, in 100 nontyphoid cases, he obtained a positive result once.

I. I. Lemann[2] reports his results in the examination of **188 specimens of blood from healthy persons,** or from convalescents from, or those sick with, other diseases than typhoid fever. Only 2 of these 188 cases gave a positive reaction—an error of 1.06 %; in one of these cases a second examination could not be made, and in the other a second examination was negative. Both cases were nonfebrile, and both patients denied having previously had typhoid fever. The author particularly directs attention to the fact that among those examined there was a series of cases in which the **infection was probably by the colon group of organisms,** and in 3 out of 5 cases of appendicitis in this group, a colon bacterium was isolated. Hence, infection with the colon bacterium does not seem to produce a positive Widal reaction. Lemann insists upon the importance of the test and upon its reliability.

Marsden[3] states that of 214 cases of typhoid examined by the Widal reaction, 209 showed a reaction in accordance with the clinical symptoms.

H. D. Rolleston,[4] in a note subsequent to his paper, states that the agglutination reaction was used in a large number of cases of typhoid fever in South Africa, but gave positive results in only 64.5 % of the cases. He considers it possible that these **unsatisfactory results were due to the use in the test of a strain of culture different from that with which the patient had been infected.** He thinks that the failure of inoculation to protect absolutely may be due to the same cause.

R. J. Cole[5] studied 15 cases of typhoid fever, in order to determine the

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[1] Giorn. med. del regio esercito, 1901, No. 5.

[2] New Orleans M. and S. Jour., Oct., 1901, p. 233.

[3] Lancet, July 13, 1901.

[4] Brit. Med. Jour., Oct. 12, 1901.

[5] Bull. Johns Hopkins Hosp., July, 1901.

frequency of typhoid bacilli in the blood. The blood was removed from an arm vein with careful antiseptic precautions. In 11 of the 15 cases the bacilli were found, once as early as the sixth day. In 5 of the cases the cultures were positive, while the serum-reaction was still negative. In half the cases in which they were looked for, bacilli were found in the urine. The diagnosis can be made by blood cultures, in case the result is positive, in about 36 hours. In some cases the cultures were positive when the other diagnostic measures were negative.

R. Polacco and E. Gemelli[1] report their study of 50 cases of typhoid fever for the presence of **typhoid bacilli in the spots,** and state that in each instance they had positive results. The diagnosis was confirmed by necropsy, by the further clinical course, or by the later appearance of the Widal reaction. They insist that liquid culture-medium should be used, solid mediums usually giving negative results. They also always inoculate from more than one spot, and state that some of the tissue should be used in the inoculations. The instrument which they used is the vaccinostyle Maréchal. They believe that the **bacillus is always present in the spots,** and that this is a **rapid and satisfactory method of making a diagnosis.** They are now investigating the reason for the presence of the typhoid bacillus in the spots; they do not agree with Neufeld that it is the result of embolism.

Strada and Pasini,[2] in discussing the **clinical value of Piorkowski's method** for the determination of the presence of the typhoid bacillus, state that they believe that a **certain diagnosis may usually be reached within 24 hours.** They insist upon the difficulty of keeping the temperature at 22° C. (71.6° F.), and, in order to overcome this difficulty, they recommend the addition of 0.3 % to 0.4 % of agar, when the plates may be kept at a temperature as high as 26° C. (78.8° F.) without liquefying. The alkalinity also should not be so marked that, after adding gelatin, the medium is alkaline, or even neutral. The differential characteristics, by this method, are much more marked in fresh cultures, made directly from a patient, than they are in old laboratory cultures.

Bayet[3] discusses the clinical **relations between appendicitis and typhoid fever,** directing attention to the great similarity that these conditions may bear to each other in their early stages. The examination of the blood he considers of much importance, particularly counting the leukocytes and the investigation of the clotting of the blood. The fibrin elements are present in large amounts in appendicitis; the clot contains little fibrin in typhoid fever. Serum diagnosis is the most important method of diagnosis, but nevertheless it frequently fails. If the diagnosis is thought to be appendicitis, the author believes that operation should be undertaken, even though some question remains as to the nature of the case, as he considers that more lives would be saved in this way than by waiting.

R. Mühsam,[4] in discussing the differential diagnosis of appendicitis and typhoid fever, reports a case in which the **illness began with a chill,** which is unusual in typhoid, and with **severe pain in the ileocecal region.** There was also a tender area of resistance about McBurney's point. The patient had none of the appearances of typhoid fever; spots and splenic enlargement were absent. Operation was undertaken in the belief that it was a case of appendicitis, and a typhoid ulcer of the cecum was found, evidently on the verge of perforation. This was folded in, the serous surfaces were brought together over it, and the patient recovered from the operation without any unfavorable symptoms. No other ulcers were discovered, but the mesenteric glands were enlarged. The subsequent course of the case was entirely satisfactory, and the patient ultimately recovered. The diazo reaction never appeared; the Widal reaction appeared first on the eighteenth day; and Piorkowsky's reaction was obtained 12 days after the operation.

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[1] Centralbl. f. innere Med., Feb. 1, 1902.

[2] Gaz. degli osped., No. 6, 1901.

[3] Gaz. des Hôp., No. 85, 1901.

[4] Deut. med. Woch., No. 32, 1901.

E. Schwartzkopf[1] describes a case of **severe epidemic parotitis** with unusually grave symptoms. The case in some particulars resembled one of typhoid fever, and the diagnosis was at first rather difficult. The patient, a man of 29, had at first pain and swelling of the parotid gland, associated with chill, marked fever, severe headache, vertigo, and profound weakness. He became somnolent and apathetic, and had marked diarrhea. His appearance was very much that of a case of typhoid fever, or some other severe general infection. The liver and spleen were tender upon pressure over them, but could not be palpated; the diazo reaction was negative and the Widal reaction was apparently not tested. There was some pain in the region of the left testicle, but the testicle did not swell. The fever soon declined and reached normal on the sixth day of the disease. The difficulty of diagnosis as concerned typhoid fever was most marked in the early part of the attack. A diagnosis of mumps was made because of the existence at that time of an epidemic of mumps in the town.

Treatment.—U. F. Martin[2] describes a very **simple frame,** which can be readily applied to the bed and used **for a typhoid slush bath.** It consists of only 4 pieces—2 end-pieces and 2 foot-bars. It is used by placing a rubber sheet and a blanket under the patient, applying the frame to the bed, and lifting the rubber sheet over the bars and hooking it on by loops of tape. The water can readily be run out of the bath into a tub afterward.

A. E. Wright[3] discusses the results obtained by **antityphoid inoculation** in an epidemic of typhoid fever which occurred in an asylum in Dublin, 54 cases of the disease having occurred. The percentage of cases occurring in the uninoculated was 10.1; and in the inoculated, 1.5.

P. Selby[4] reports upon his use of **whey instead of milk** in typhoid fever, and insists that the results from the use of whey were far more satisfactory than those obtained with milk.

Sacker[5] discusses the **use of gelatin and its effect upon blood coagulation.** He states that intravenous and subcutaneous gelatin injections produce changes in the blood-vessels, and that under the microscope one can observe constant changes in the blood, chiefly consisting in agglutination of the corpuscles. He believes, therefore, that gelatin injections—in animals, at least—cannot be looked upon as without danger. He has seen embolism and secondary thrombosis occur in the blood-vessels of living animals, as the result of an increased tendency to intravascular coagulation. In human beings the increased tendency to coagulation is principally active in those portions of the blood-current in which the blood-stream is slow, and in which there are alterations of the vessel-walls; hence, in aneurysm there should be a general tendency to healing of the aneurysm. The author thinks, therefore, that the use of gelatin in hemorrhage or aneurysm is justified when other measures fail, but that it must be looked upon as a dangerous method of treatment.

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[1] Deut. med. Woch., Oct. 17, 1901.

[2] Med. Rec., Dec. 2, 1901.

[3] Brit. Med. Jour., Oct. 26, 1901.

[4] Lancet, Nov. 2, 1901.

[5] Mittheil. a. d. Grenzgeb. d. Med. u. Chir., Bd. VIII, Hefte 1 und 2.

E. Levy and H. Bruns[1] report that they have **investigated 6 specimens of commercial gelatin, for the tetanus bacillus,** using the method of SanFelice and making cultures. Of the gelatin, 2 or 3 grams were dissolved in 100 cc. of bouillon, allowed to stand for 8 or 10 days in an incubator, and filtered through a Pasteur-Chamberland filter; and the filtrate was injected, in quantities of 0.03 to 0.04 cc., into mice. **In 4 of the 6 specimens they demonstrated the presence of tetanus bacilli.** These may also be found in the unfiltered culture-fluid; but it is unsatisfactory to inject this into the animals, because other bacteria which are present (malignant edema, etc.) often cause death within 24 hours. It has generally been stated that tetanus spores may be killed by steam at 100° C. (212° F.) in 8 minutes. The authors have not found this to be true, and consider it possible that different tetanus bacilli may have varying degrees of resistance to heat. It is not possible to use high temperatures with gelatin for a prolonged period, without destroying its gelatinizing powers; and it is important to discover whether any hemostatic action that gelatin may possess is interfered with by prolonged heating.

Schmiedicke[2] also reports that in examining commercial gelatin for the presence of tetanus bacilli he has had positive results, the number of specimens examined and the frequency of the positive results not being stated. The method he used is the simple one recommended by Heyse; tubes are filled with agar to a height of about 10 cm.; this is liquefied and cooled to about 40° C. (104° F.), and pieces of gelatin about 2 cm. square are dropped in and allowed to sink to the bottom. After about 5 or 6 days the colonies which develop in the depths are investigated further.

Racchi[3] is referred to as recommending the use of **gelatin by rectal enema,** considering that its action is much more rapid in this way than when used hypodermatically.

J. A. Tschuschner[4] states that he has used **gelatin and gluten internally** in a number of cases of gastric, intestinal, and uterine hemorrhage, with markedly hemostatic results.

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[1] Deut. med. Woch., Feb. 20, 1902.

[2] Deut. med. Woch., Mar. 13, 1902.

[3] Practitioner, Nov., 1901.

[4] Prag. med. Woch., No. 19, 1902.

MALARIAL FEVER.

Etiology.—J. H. Egbert,[1] in discussing malarial fever in Central America, insists that **fleas transport the disease,** and that measures which do away with the fleas are sufficient to decrease the number of cases of malaria decidedly.

Henry B. Orr[2] **describes a parasite of the common night mosquito,** Culex pungens, which, while not related to malaria, is of interest in relation to the question of transmission of disease by the mosquito. The parasite was found very frequently in mosquitos captured in the center of the city. It is somewhat pear-shaped, with a glassy, greenish body, filled with fine granules, and having a nucleus; it has a clear, hyaline pseudopod. A second form is bacillus-like in structure, and there is a third form, which resembles the first, but has a cilium instead of a pseudopod. There is also a fourth type, which resembles the third, except that, in addition to the cilium, it has a thick, hyaline tentacle, springing from the under side of the body. The parasites are capable of living—for a short time, at least—in the digestive juices, in blood, and in rain-water. It is not believed by the author that they are pathogenic; but his experiments show the possibility of pathogenic parasites being carried by the Culex, and of their producing intestinal derangement, for they could pass the stomach without digestion.

P. T. Manson[3] gives **a further report of his own case of malarial infection,** which was previously reported as having been experimentally produced by using infected mosquitos. His first attack occurred in September, 1900. He was apparently rapidly cured by quinin. On May 30, 1901, **9 months later,** while living in Aberdeen, he had **another typical attack of tertian malaria,** with the typical parasites in the blood.

A. F. A. King[4] considers that **light,** and red light in particular, **aids the development of the malarial plasmodium;** while purple or violet light and fluorescence, which intensifies the violet rays, reduce its activity. He recommends that malarial patients be kept in the dark or in a purple or blue light, and that their garments be impenetrable to light.

Symptomatology and Complications.—C. F. Craig[5] discusses **latent and masked malarial fever.** He describes 195 cases, in addition to the 172 cases that he has previously reported. These occurred in soldiers returning from the Philippine Islands, and were discovered in 15 months in the routine examination of the blood, every patient admitted to the Presidio at San Francisco being subjected to a routine blood examination; this having shown many malarial infections that would otherwise have been undiscovered, and in many instances having saved lives. There were **44 cases of tertian infection, 1 of quartan, and 150 of estivo-autumnal.** It has been generally stated that tertian infection is the most common in the Philippines; but the author is inclined to believe that this statement is made because estivoautumnal infections have been overlooked. In the tertian infections, pigmented parasites were generally present, and were quickly and easily recognized; in the estivoautumnal form the hyaline ring-form was the most common. Hence these could be readily overlooked. Crescentic forms were observed only 12 times in 150 estivoautumnal infections. The number of parasites was, in most instances, noted as being small. In many cases it was difficult to account for the lack of clinical symptoms. The diagnoses in the cases given before the malaria was discovered are reported. In the so-called latent cases, in which the disease caused no noteworthy symptoms, the **most common symptoms were those of chronic diarrhea.** In the masked cases 55 were called chronic dysentery, 14 pulmonary tuberculosis, and small numbers were given other names. Of the total number, 90 were masked infections; 105, latent. In the cases of dysentery the presence of malaria could not have been discovered without the blood examination. All the symptoms could have been explained through the diagnosis of dysentery. Of the 195 cases, 180 showed a definite source of the infection; of these, 120 had the first attack in the Philippine Islands, while 60 had their first attack in Cuba. Five patients died. The postmortem conditions are to be discussed at length in a later paper.

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[1] Med. Rec., Aug. 17, 1901.
[2] New Orleans M. and S. Jour., Dec., 1901.
[3] Brit Med. Jour., July 15, 1901.
[4] Am Jour. Med. Sci., Feb. and June, 1902.
[5] Med. Rec., Feb. 15, 1902.

J. M. Jackson[1] reports a series of cases of malaria in which there were **very acute abdominal symptoms,** in several instances, at least, warranting the diagnosis of peritonitis or appendicitis. One case was unusual in the fact that severe abdominal cramps preceded the onset of the chill by about 3 weeks.

J. T. Moore,[2] in a study of **nephritis in malarial fever,** concludes that a double tertian infection produces nephritis in a very large percentage of cases, even when it runs only a short time; while in a simple infection nephritis is uncommon, if the course is short. The more chronic the case, however, the more liable is the patient to nephritis. The estivoautumnal type probably produces the greatest number of cases of nephritis.

J. Ewing[3] reports the case of a girl of 17 who was taken with jaundice and pallor, followed by chills and fever; enlargement of the spleen; some edema; and the presence of albumin, epithelial cells, and blood-cells in the urine. The diagnosis was thought to be typhoid fever with acute nephritis. Autopsy showed **acute hemorrhagic nephritis.** Microscopic examination demonstrated that there was a tremendous **massing of malarial parasites in the kidneys.** The glomeruli exhibited pigmented cells and some parasites within the capillaries. The lining cells of the convoluted tubules showed extreme swelling and degeneration. The parasites were small and deeply pigmented, and had the appearance of fully grown estivoautumnal parasites.

Pansini[4] discusses a **cerebellar syndrome** which occurred **in 4 cases of acute malaria.** The symptoms consisted in relaxation of the whole body, the patients at first being unable even to sit up in bed; inability to carry out any purposeful movement, and absolute inability to walk. Later on, the patients could maintain their equilibrium only with difficulty, exhibiting the drunken gait of cerebellar subjects. There was, however, no paralysis; it was merely an asthenia. Speech was impossible or extremely difficult. There was no muscular atrophy, no reaction of degeneration, and no disturbance of the skin sensation or of the muscle sense. The reflexes were normal, as were the sphincters. There was practically no psychic disturbance. The conclusion reached was that there was lesion of the peripheral nerves, of the medulla, or of the cerebrum. The chief symptoms were **loss of equilibrium, muscular asthenia, and complete ataxia.** The production of these cerebellar symptoms is referred to the disturbance of circulation due to the deposition of pigment in the small vessels. The absence of all signs of irritation or of compression of the medulla speaks strongly against hemorrhage. The occurrence of these symptoms emphasizes the necessity for very active intervention in cases of pernicious malaria, and for the use of specific treatment long after the attack has passed off. The symptoms last for a long time, and the prognosis concerning absolute recovery should be very carefully stated in such cases.

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[1] Boston M. and S. Jour., June 26, 1902.

[2] Amer. Med., Dec. 28, 1901.

[3] Am. Jour. Med. Sci., Oct., 1901.

[4] Riforma med., Nos. 264 to 268, 1901.

J. B. McElroy[1] reports the case of a negro of 60 who had repeatedly had malarial infection, and who had had paroxysms of fever for 4 days previous to February 11th, when he had a severe chill followed by delirium and coma, the latter persisting 7 hours. When seen, he had **large bullæ over both knees,** and smaller ones over the middle of the leg. These bullæ looked like burns. The malaria subsided under treatment, but the bullous areas became gangrenous and caused extensive ulcers after sloughing. The author mentions 2 differential counts in pernicious malaria. In one case the result was: polymorphonuclears, 64 %; large mononuclears, 20 %; small mononuclears, 16 %. The second showed the same count, except that 1 % of the large mononuclears was replaced by eosinophiles. McElroy insists that the negro is not immune to malaria, and mentions 20 cases of pernicious fever which he has recently seen, and states that 60 % **of these were in negroes,** and that the latter exhibited a mortality of 33.5 %; the whites, one of 25 %. The case of gangrene especially reported occurred in an estivoautumnal infection. The author mentions another case, probably one of malarial gangrene, which occurred in a mulatto girl of 4 years. In this case spontaneous amputation occurred at the upper third of the leg. In discussion V. C. Vaughan said that pernicious malaria was as common among negro soldiers at Santiago as it was among the whites.

Gremillion[2] reports a case of **polyneuritis** in a patient with enlarged liver and spleen who had often had malarial attacks. He recovered entirely. There was no examination of the blood.

M. Otto[3] describes a case of **quartan malarial fever which was complicated by hemoglobinuria.** The case was of much interest because of the rarity of black-water fever with quartan malaria and because the **patient had never been out of northern Europe.** He had had malaria 24 years before, in Cracow, but had lived for 18 years in Hamburg, Germany. The case was also of decided importance because it clearly demonstrated the relation of quinin to the hemoglobinuria. The man had the first symptoms of malaria in the middle of August. His disease was not recognized, and quinin was not given until September 13th. An hour and a half after taking 7½ grains of the drug he had pains in the hips and extremities, severe gastric disturbance, and a marked sense of anxiety; a few hours later he passed very deeply pigmented urine. Some weeks later the actual malarial nature of his disease was recognized by the author, and small doses of quinin were given by the rectum. The man soon recovered. A dose of 15 grains of quinin was subsequently given, to test its effect upon the urine. The patient at once had a marked, although milder, attack of hemoglobinuria.

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[1] Jour. Am. Med. Assoc., Sept. 14, 1901.

[2] New Orleans M. and S. Jour., Aug., 1901.

[3] Deut. med. Woch., Jan. 23, 1902.

F. K. Kleine[1] reports **15 cases of malarial hemoglobinuria,** to demonstrate the extremely close relation that quinin bears to the production of this condition. In each instance the hemoglobinuria followed more or less closely upon the administration of this drug; and the author considers that the more carefully records are studied, the closer will this relation be found to be. The reason that black-water fever so rarely occurs in northern latitudes is chiefly, he thinks, that so extensive and unregulated a use of quinin as that common in the tropics is never seen with us; but, with Koch, he believes that the appropriate use of the drug as a prophylactic will be sufficient, in the vast majority of cases, to overcome the malaria and also the tendency to black-water fever, for quinin acts only on corpuscles that are already damaged by malaria.

Fiocca[2] notes that the **coincidence of typhoid fever and malaria** has not been demonstrated in the malarial regions of Italy. Cases of this kind have been reported, but their true nature has not been satisfactorily shown. The author describes a case in which he has demonstrated that both diseases were present in the patient at the same time. The man had had malaria, and was taken ill with typhoid fever in the latter part of November. During this time malarial parasites were entirely absent. After the typhoid-fever curve had subsided, the malarial attacks reappeared. There was then a characteristic typhoid relapse, and during this time malarial parasites were absent from the blood. With the appearance of the typhoid infection, therefore, in both instances, the evidences of the malarial infection disappeared. The possibility of malarial infection in the hospital was excluded.

Diagnosis.—Grixoni[3] claims that when 2 drops of malarial blood are allowed to flow together beneath a cover-glass, the **red cells approach each other, group themselves together, and form small clumps,** within about a minute. They sometimes remain normal in appearance, or they may be dissolved or lose their hemoglobin. The clumps are composed of from 5 to 100 cells, and all the red cells become clumped, the leukocytes remaining unaffected. Malarial blood has the same action on normal blood and on blood from cases of other diseases. **Quinin interferes with the agglutination.** The phenomenon was not observed in other diseases, except typhoid fever. The persistence of this phenomenon indicates that the malaria is not cured.

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[1] Brit. Med. Jour., Sept. 14, 1901.

[2] Il Policlinico, Sept., 1901.

[3] Gaz. degli osped., No. 57, 1901.

Prophylaxis.—Laveran,[1] in reporting for the commission appointed to recommend methods of **prophylaxis against malaria in Corsica,** first refers to the fact that the prevalence of that disease has largely depopulated considerable portions of the island, through sickness and the inability to carry on life there. He states that he has found the Anopheles in large numbers in Corsica. As to prophylaxis, he recommends the prophylactic administration of quinin; but this would necessitate the gratuitous distribution of the drug, as the people are usually too poor to pay for it. There should also be attempts made to destroy the mosquitos, a procedure which, he thinks, has been somewhat successfully accomplished in other regions; and mechanical measures of protection against these insects should be introduced.

G. Nuttall[2] has made an investigation of the **colors which attract and repel Anopheles,** and describes a color-scale, in relation to its attractiveness for this insect, which reads as follows: Marine blue, dark red, and black are the most attractive; less attractive are dark green and violet blue; much less so are pearl gray, light blue, white, and orange; yellow does not attract them at all. The observations are of value as indicating that yellow clothing, and the use of yellow colors about dwellings, will serve as some protection against Anopheles. The use of the khaki uniform in the tropics is, consequently, rational from this standpoint as well as others.

W. R. Battey[3] states that in some experiments on killing mosquitos in a stone receptacle for rain-water, after having unsuccessfully tried kerosene oil, he **used eucalyptus oil, and found that this rapidly exterminated the insects.** The latter oil is, however, too expensive for widespread use.

Sergent[4] describes a region on the banks of the Yonne, a tributary of the Seine, in which **Anopheles is found in large numbers, and yet in which malaria has disappeared.** He considers the presence of the mosquitos, however, to be an element of marked danger to the population; for numerous persons infected with malaria return to this region from the colonies, and are likely to have new attacks. The mosquito can then readily carry the infection to others.

Treatment.—A. Merkel,[5] in a study of the **excretion of the metabolism products of quinin,** states that in the dog all but 12 % or 14 % of quinin is destroyed in the organism, the destruction not being less at the beginning of the use of the drug than 4 weeks later. The remainder appears in the urine as a basic product of quinin. The chief conclusion for clinical purposes to be derived from this is that **if an immediate**

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[1] Bull. de l'acad. de méd., Dec. 24, 1901. [2] Brit. Med. Jour., Sept. 14, 1901.
[3] Brit. Med. Jour., Sept. 28, 1901. [4] Ann. de l'Inst. Pasteur, Oct., 1901.
[5] Arch. f. exper. Path. u. Pharm., Bd. XLVII, Hefte 3 u 4.

effect is desired, doses of the drug must be large, and may better be given subcutaneously.

C. J. Gremillion[1] describes a case of **remittent malaria** with a marked **idiosyncrasy to quinin,** in which he used very **large doses of Fowler's solution,** giving as much as 5 drops every hour for over 2 days. The patient recovered satisfactorily.

C. B. Ferguson[2] strongly recommends the **hypodermic use of quinin** in malarial fever, even in cases that are not malignant. After investigating a series of salts of quinin, he found that the most satisfactory is the bi- (or acid) hydrobromate. This dissolves in 6 parts of water and is readily absorbed. The injections are practically painless.

M. J. Rosenau,[3] investigating the **destruction of mosquitos by disinfectants,** found that formaldehyd is very ineffective unless used in enormous quantities and liberated very quickly. Sulfur dioxid is an excellent insecticide, and the author considers it the disinfectant of the first rank in proceeding against yellow fever, malaria, filariasis, and other diseases carried by insects.

Malaria and Carcinoma.—Löffler[4] suggests, under the title of a **new treatment for carcinoma, infection of carcinomatous patients with malaria.** He draws attention to the fact that in regions in Europe where malaria has largely disappeared carcinoma has increased, while carcinoma is very uncommon in the tropics, where malaria is rife. Other infections seem at times to have had good influence upon carcinoma, and Löffler thinks it is worth while to try this treatment.

Spitzly,[5] in discussing the **relation between carcinoma and malaria,** states that during his 8 years' service in Surinam he found malaria extremely common; and that he observed that many persons who had malaria, or who had had the disease, developed carcinoma of various regions. The occurrence, the rapidity of growth, and the development of metastasis seem to be uninfluenced by malaria.

Prochnik,[6] in discussing Löffler's suggestion that malaria may be inimical to carcinoma, states that his observations in the tropics lead him to the definite conclusion that **carcinoma is not uncommon in the colonies** in either Europeans or natives, and that malaria has no evident influence in preventing carcinoma. He also states that the fatalities recorded from carcinoma in the tropics are largely instances of carcinoma of the liver. Malaria is so common in the tropics that one may take it for granted that practically every person who develops carcinoma has already had malaria. The author considers that malaria is probably not transmitted exclusively by mosquitos.

H. Breitenstein,[7] in referring to the relation between carcinoma and malaria, states that in Borneo he observed **6 cases of carcinoma, in a region in which malaria is very common.** He considers Löffler's theory extremely questionable.

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[1] New Orleans M. and S. Jour., Aug., 1901.

[2] Brit. Med. Jour., Feb. 22, 1902.

[3] No. 6, Hygienic Laboratory of the U. S. Marine Hosp. Service.

[4] Deut. med. Woch., Oct. 17, 1901.

[5] Brit. Med. Jour., Jan 4, 1902.

[6] Wien. klin. Woch., No. 5, 1902.

[7] Prag. med. Woch., No. 45, 1901.

YELLOW FEVER.

Etiology.—W. Reed and J. Carroll[1] have undertaken further **studies of the etiology of yellow fever, after the manner of those carried out by Löffler and Frosch concerning foot-and-mouth disease.** The latter showed that filtered lymph causes foot-and-mouth disease as promptly as unfiltered, and concluded that the specific agent of the disease is so minute as to pass through a porcelain filter. Reed and Carroll produced cases of yellow fever by allowing infected mosquitos to bite human subjects. Six persons were so bitten, and 2 acquired the disease. The authors used (*a*) unheated and partially defibrinated blood, (*b*) partially defibrinated blood heated to 55° C. (131° F.), and (*c*) diluted blood-serum that had been filtered through a Berkefeld filter. One person was inoculated with the unheated and partially defibrinated blood, and exhibited a mild attack of yellow fever. The partially defibrinated blood heated for 10 minutes at 55° C. was negative in 3 persons; hence, this temperature must destroy the specific agent of the disease. The diluted and filtered serum was used in 3 cases; two passed through yellow fever, and the other had a brief rise of temperature. The filter which had been used was then tested, and was shown to be, in the ordinary sense, capable of removing bacteria. The infection was apparently not due to the mere presence of toxin in the filtered serum, as shown by the results of heating. As a further test, the authors transferred the blood of one of the patients that had been infected by filtered serum to a third person, and produced yellow fever in this individual. They **believe, therefore, that the specific agent of the disease passes through a porcelain filter.**

W. Reed[2] gives a general discussion concerning the **propagation of yellow fever,** based upon recent researches, going over the work regarding infection with mosquitos. He gives a table showing the result in 16 persons, 11 being positive on the first infection with mosquitos, 1 negative, 1 negative on blood injection, and one of the cases infected with mosquitos having been negative on attempted infection with blood. He also considers the results of study elsewhere in relation to the ordinary period of development of the disease. At Orwood and Taylor, in Mississippi, during 1898, Carter had found the period of incubation to vary from 11½ to 29 days; that is, it corresponded with the interval observed after infection with mosquitos. From personal experience, Reed adds the cases of 2 nurses who had attended yellow fever cases; one sickened on the fourteenth, and the other on the fifteenth day. He believes that the period of **13 to 23 days indicates the ordinary time of incubation.**

Sanarelli[3] contributes an article concerning yellow fever, in which he insists that it is **impossible to explain the infection in this disease through the medium of insects.** He admits that insects may occasionally carry the infection, but thinks that they have only a secondary importance. He also criticizes the work of Durham and Meyers on the bacteriology of the disease.

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[1] Amer. Med., Feb. 22, 1902.

[2] Med. Rec., Aug. 10, 1901.

[3] Gaz. degli osped., 1901, No. 102.

The Commission on Mosquito Infection of the Orleans Parish Medical Society, consisting of G. E. Beyer, O. L. Pothier, M. Couret, and I. I. Lemann,[1] to report an **extensive investigation into the relation of mosquitos to disease,** summarizing their work in the statement that they have found that Anopheles inhabits the swamps surrounding the city of New Orleans and the outskirts; while Stegomyia and some of the more common forms of Culex are found in the heart of the city, breeding in cisterns and gutters. There is an interesting relation between the location in which these mosquitos were found and the regions in which yellow fever occurred in 1897; but the facts are not sufficient for the Commission to lay emphasis upon this point. They believe that Bacillus icteroides is a distinctly pathogenic organism. They have infected Stegomyiæ fasciatæ by allowing them to bite animals infected with Bacillus icteroides, and suffering from a disease corresponding experimentally with yellow fever as observed in animals. **From these infected mosquitos they recovered Bacillus icteroides in pure culture.** They also produced the same disease in healthy animals by allowing them to be bitten by mosquitos infected from 5 to 11 days previously, and by inoculating others with cultures made from infected mosquitos. These bacilli were recovered from the mosquito-infected animals. So far, the Commission has been unable to locate and stain the bacilli in the tissues of the mosquito. They have confirmed previous investigations concerning the presence of the malarial parasites in Anopheles, and the life-cycle of the plasmodium in the body of the intermediate host. Epidemiologic considerations make them believe that **Anopheles maculipennis produces simple tertian and quartan infections,** while **Anopheles crucians produces the estivoautumnal disease.** In prophylaxis, they insist upon the importance of getting rid of cisterns as fast as possible, of abolishing surface drainage, and of substituting sewerage for privy vaults. Meanwhile, cisterns should be screened, gutters flushed, oil poured on privy vaults and stagnant pools, and all persons suffering from malaria or yellow fever screened by mosquito-netting, in order to prevent the access of mosquitos.

Treatment: Prophylaxis.—W. Reed and J. Carroll,[2] in discussing the prevention of yellow fever, give an extensive description of Stegomyia fasciata, formerly known as Culex fasciatus, the insect which seems to transmit yellow fever. They state that **infected boxes, trunks, etc., cannot transmit the disease,** as the mosquitos die within a few days when deprived of water, all of these insects perishing within 5 or 6 days. **Five days is, then, a satisfactory period of observation of vessels** which are suspected of infection. Prevention can be accomplished by **screening the windows and doors of a room occupied by a patient and destroying all the mosquitos in the house.** An exposure to the fumes of sulfur for an hour and a half, using 1 pound of sulfur to 1000 cubic feet of air-space, is sufficient to destroy all mosquitos; formaldehyd gas is not so efficient; pyrethrum powder, in the proportion of 4 ounces to 1000 cubic feet of air-space, will stupefy the mosquitos, and they may then be swept up and burned. In the treatment of vessels and of their passengers and cargoes, a distinction should be made between infected and noninfected ports; and in the case of vessels sailing from infected ports, between those that have received their cargoes and passengers in mid-stream and those that have loaded at the wharf. The authors think that no quarantine restrictions should be imposed upon either passengers or cargoes from noninfected ports. If the vessel is loaded in mid-stream, infection can be conveyed only by passengers who have been on shore. In the case of such vessels the nonimmunes should be quarantined for 5 days, counting the time consumed by the voyage; and the vessel should be allowed to discharge her cargo without delay. If there has been sickness on board, the sick should be removed, the forecastle and staterooms should be disinfected with sulfur or formaldehyd, and the vessel should be allowed to proceed to her wharf. If the vessel has received her cargo at the wharf of an infected port, there may have been infection through contaminated mosquitos or through some individual who has acquired infection on shore. If from 16 to 21 days have not elapsed between her departure from the port of sailing and her arrival at a United States port, there is no way of absolutely excluding the possibility of the importation of the disease, except by the detention of all nonimmunes for a sufficient number of days to show their freedom from infection and, at the same time, carefully disinfecting the quarters of the crew and passengers. If more than 20 days have passed during the voyage, there is no reason why either passengers or ship should be detained. The authors do not think that it is necessary to disinfect the cargo.

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[1] New Orleans M. and S. Jour., Jan., 1902.

[2] Med. Rec., Oct. 26, 1901.

W. C. Gorgas,[1] in discussing the work of the Sanitary Department of Havana, and particularly referring to the repression of yellow fever, states that the present method of treating a case that has been notified is to place the patient in any room of the house that may be chosen, to fit the doors and windows of this room with wire screens, to place a guard about the house to keep out unauthorized persons, and to insist upon care in the management of the case, particularly in regard to mosquitos. **Pyrethrum powder is then burned in the various rooms of the house;** and, at the end of 3 hours, the mosquitos are swept up and destroyed. Three or four contiguous houses are also treated with the powder. The author gives a table of the number of cases of the disease which have occurred in Havana in recent years. The **methods of combating it have apparently been successful;** for, up to the end of July, 1901, but 2 deaths had occurred. In 1899, 6 deaths occurred in the same period; but, with this exception, there had been, since 1890, from 44 to 531 deaths during the same period. The number of cases occurring after the war on mosquitos was begun was also extremely small; and the city was absolutely free from infection from March 8th to April 20th, and from May 7th to July 1st.

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[1] Med. Rec., Sept. 7, 1901.

VARIOLA.

A. MacGugan[1] discusses an **epidemic of smallpox** at the Michigan Asylum for the Insane, and mentions 2 cases of **relapsing variola.** Other cases like these, from the practice of De Lano, are referred to. [In cases with such mild first attacks it is exceedingly important to determine that the first attack was smallpox, and that the second attack was not due to exposure to the disease through erroneous diagnosis and isolation with other cases of smallpox.] The author mentions 2 cases of **variola without eruption.** Chills occurred in only 13 of the 50 cases. Fever was marked, however, in many of the mild cases. Headache was always present, and diffuse pains occurred in 90 %. Nausea occurred in only 14 cases. With only one exception, there was practically no delirium in any of the cases. In varioloid the onset, contrary to the usual teaching, was ordinarily severe. MacGugan states that an examination of the pustules in 7 cases, of the pustules of vaccinia in 2 or 3 cases, and of a few tubes of vaccine, showed the organisms described by Pfeiffer and Funck. In the 50 cases reported, **17 had been vaccinated, but none of them recently.** Of these 17 cases, only 3 had, however, severe attacks. Eight patients were vaccinated after recovery, but in no instance did the vaccination take. The diazo reaction was looked for in a number of instances with negative results.

T. J. Happel,[2] in a further discussion of what he has **termed pseudo or modified smallpox,** states that since his previous report **about 100 cases** have come under his observation, and have confirmed his doubts as to the possibility of the epidemic being true variola. He insists upon the frequency with which the disease occurs among the vaccinated and upon its very great mildness. In one town in his county, in which there were 600 or 700 inhabitants, 100 cases occurred, with only 1 death; and that was in an old feeble patient, and was really thought to have been due to carbolic-acid poisoning. The author gives a table of differential diagnosis between this disease and true smallpox, the main points being that, according to his statement, in the disease which he discusses there are no varieties; that the symptom of onset is chiefly fever, with little or no vomiting; that there is an acne-like eruption on the fourth day, which almost at once becomes vesicular, the vesicles not being umbilicated; and that, with the appearance of this eruption, the temperature at once falls, and the patient feels practically well. The vesicles do not suppurate, and there is no secondary fever. The eruption does not extend into the true skin.

H. M. Bracken[3] states that in the past 2 years there have been **7211 cases of variola in Minnesota, with 49 deaths**; but he believes that other deaths from smallpox have been reported as due to other causes. He insists that in the course of the epidemic there were many typical cases, as well as the frequent atypical ones. He also insists that vaccination does definitely protect against this disease. In a series of 662 cases, only

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[1] Med. News, Dec. 14, 1901.
[2] Jour. Am. Med. Assoc., Aug. 3, 1901.
[3] Jour. Am. Med. Assoc., Aug. 3, 1901.

10 had been successfully vaccinated. Of these 10, one had been vaccinated 6 years before; others over 20 years before. Two had been vaccinated recently, but both of these had very mild attacks. The author emphasizes the fact that a modified vaccinia occurring after an attack called variola does not show that the previous disease was not variola. He thinks that a **recent vaccination is more marked in its protective effect against variola than is an old attack of the disease itself.** The duration of immunity to smallpox or vaccinia is very variable. Bracken also contends that a modified variolous eruption about the seat of a vaccination has been frequently mistaken for successful vaccination, observers overlooking the fact that the eruption of variola is likely to be confluent about the seat of any especial irritation; as, for instance, a cut or a scratch. He has been unable to find more than one case in which he believes that a successful vaccination occurred immediately after an attack of variola; and this case had vaccinoid, and not true vaccinia. He mentions, however, a number of cases which show the possibility of successful vaccination a long time after the occurrence of variola. He believes that it is an open question whether the type of variola so frequently seen recently is unusual or not. He thinks that similar epidemics have repeatedly occurred before, and states that variola at times exists in very mild form in tropical countries. In the discussion, W. Bailey said that the type of the disease met in Kentucky was undoubtedly actual smallpox, and he believed that Happel was wrong in thinking that the disease in Tennessee was not variola. L. Leroy agreed with this view, on account of his experience in Tennessee, which had been that almost all the vaccinated escaped the contagion. There was a general concurrence in this view; and a resolution was adopted, in which it was stated that the disease prevailing extensively in the United States, and in some cases called pseudo-smallpox, is genuine smallpox, and should be managed as such.

H. Spalding[1] discusses the **diagnosis of mild and irregular smallpox,** as seen in the recent outbreak in the United States. Early in March, 1899, one case of smallpox was imported into Chicago. Within 17 months after this, there were 72 cases, 25 of which were direct importations. For 3½ months after this, the city was free from the disease. A mild case was then again imported, and came into close contact with numerous persons. Since this time, there have been 238 cases, 39 of which came from without the city. No cases of smallpox without eruption were seen in this number, nor were there any cases with initial rash. There were 177 cases of mild, discrete smallpox in unvaccinated persons—a very remarkable fact. The prodromal symptoms, in many cases, were also very irregular and insignificant, nausea being the most constant except fever; persons with the pustular eruption have repeatedly been found working in factories, etc. **Secondary fever was rarely present.** The author mentions one case of confluent abortive smallpox in a patient who had been vaccinated 45 years before. One person had **variola variceloides,** showing well-developed vesicles on the first day, these disappearing after 48 hours, but the patient showing a typical smallpox eruption. The eruption was often found present in different stages at the same time. Of the 310 cases, 5 had typical old scars, but the most recent vaccination in these cases was 16 years before the attack; 271 had never been successfully vaccinated. Spalding notes the extreme difficulty in the diagnosis of many cases. It is easy **to mistake the eruption of varicella for that of variola in the negro,** because the external layer of skin in an adult negro is so thick. The author has never seen any disease resembling smallpox that is not well known and well described. One hundred patients who had recovered from the mildest form of this so-called doubtful disease were vaccinated with not one successful result, although none of them had been previously vaccinated. All the patients with smallpox were placed in the same wards in the same hospital. They showed all varieties, from hemorrhagic and confluent to the very mildest type. None of the mild cases contracted smallpox from the severe cases, which is satisfactory evidence that they all had the same disease. Spalding mentions cases in which the gravest form of smallpox was contracted from the mildest variety of the disease.

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[1] Jour. Am. Med. Assoc , Aug. 3, 1901.

H. Roger and Emil Weil[1] **discuss the initial rashes of smallpox, reporting a study of the rash in 978 cases.** The percentage of occurrence of initial rash was 18.4. Varioloid gave the smallest number; hemorrhagic, the highest (47.5 %). The frequency of the rash bore a direct relation to the severity of the disease. Of the 40 hemorrhagic cases, all died; and of these, 19 had initial rashes. Women showed initial rash somewhat more frequently than men. The authors distinguish **6 forms of rash**—erythematous, urticarial, morbilliform, scarlatiniform, purpuric, and astacoid, or lobster-like. The latter rash is practically identical with the hemorrhagic form of smallpox. It may appear in apparently benign cases, or in those that are severe from the beginning. It is practically a **definite indication that the disease will terminate fatally;** and, unlike the other forms of rash, it is general, except over the face. It is not a generalized purpura, but is a diffuse hemorrhagic suffusion, of uniform tint. Various eruptions may be present in the same patient. Of the 171 cases of initial rash, 25 showed two forms of rash. The rash **almost always precedes the specific eruption,** and commonly appears on the first or second day, disappearing in 24 or 48 hours, when the true rash appears. The purpuric rashes, like hemorrhagic eruptions in other conditions, are more persistent. In partial rashes the inguinal region is most frequently involved; then the rash is likely to affect the axilla and the mammary region. The flexures are comparatively rarely involved. The site of the initial rash may determine the earliest and most severe seat of the true eruption. The authors have seen but 3 cases of late rash, all occurring after the fifteenth day, 1 being erythematous and 2 purpuric, and all the patients dying. The more general the eruption, the graver the prognosis attending it. Localized initial rashes are commonly associated with mild cases.

Von Ortynski[2] describes a case of **generalized vaccinia** in a soldier who, 10 days after revaccination, was taken with a chill and headache. Two days later a measles-like eruption appeared; and 2 days after this he showed typical pox over the whole skin surface. The fever persisted for 2 weeks, the pustules beginning to dry 5 days after their appearance, and this process being completed at the end of 15 days. The healing of the pustules was associated with the production of scars. The incubation period and the whole clinical picture were exactly like those of smallpox. Numerous other persons were vaccinated with the same lymph, without showing any unusual signs.

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[1] La Presse méd., July 31, 1901.

[2] Wien. med. Woch., No. 39, 1901.

RHEUMATISM.

Bernstein,[1] in writing of the **relation between acute articular rheumatism and trauma,** discusses trauma and its effects as follows: Infected wounds of the joints frequently cause pyemia, but rarely cause articular rheumatism; infected skin wounds frequently cause pyemia, but only infrequently produce rheumatism; subcutaneous injuries very rarely cause pyemia, but very frequently cause rheumatism. He considers that it is not at all uncommon to see an acute articular rheumatism **involving first a joint which has previously been subjected to trauma,** or involving the joint nearest to the situation of a previous trauma. It is important in studying the relation of trauma to rheumatism to exclude those cases in which the patient had previously been subject to rheumatism, as it is then difficult to decide whether or not the trauma had anything to do with the attack following it. It is also essential that the attack of rheumatism should follow the trauma within a reasonably short time. He describes a **series of 7 cases** in which he believes that trauma actually brought on an attack of rheumatism. For instance, in one case a man fell upon his left knee; within 24 hours that knee swelled rapidly, and acute swellings in other joints followed. In another case a man fell down a flight of stairs and struck his right knee repeatedly in falling. This articulation swelled within 24 hours, and this was followed by swelling in other joints. The other cases showed somewhat similar clinical histories.

McCall[2] reports an interesting case in a girl of 16, in which **rheumatism was suddenly followed by severe chorea,** and there was then a marked **eruption of rheumatic nodules,** those about the head and neck being as large as hazelnuts, while smaller ones were widely scattered. The nodules disappeared within a month. After 2 months' treatment the patient had recovered. Three weeks later, however, she again had mild choreiform movements of the right side; and after their appearance there was a new eruption of rheumatic nodules, though these were smaller in both size and number. Endocarditis also occurred during this attack.

A. Hess[3] notes that practically all writers, with the exception of Pribram, consider **venous thrombosis in acute articular rheumatism** to be rare. He reports 2 cases, both of which were interesting; the first, because of the occurrence of thrombosis of the veins of both arms and of the inferior vena cava and the iliacs, with recovery; the second, also because of the wide extent of the thrombosis, which involved the veins affected in the first case and also the superior vena cava. The second patient died. There were **curious convulsive attacks,** with temporary cessation of respiration and heart-action, which were thought to be due to the severe cardiac disturbance or to carbonic-acid intoxication, but which the postmortem showed to be probably due to the involvement of the vagus and to thrombosis in the neck. Microscopic examination showed inflammation of the vessel-walls and round-cell infiltration around the vessels. Microscopic examination of the vagus is not mentioned.

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[1] Zeit. f. klin. Med., Bd. XLII, H. 5–6.

[2] Brit. Med. Jour., Feb. 15, 1902.

[3] Deut. med. Woch., June 26, 1902.

J. Schnitzler[1] reports 3 cases in which there was a **recurrence of rheumatic joint swellings in the course of acute suppurative processes.** In all 3 cases the patients had had rheumatism some time before, and with the occurrence of localized suppuration, showed fresh joint-pains and swellings; in 2 cases these were in joints that had previously been involved, while in the third case other joints also were affected. The general condition was good, there were no chills, and the condition rapidly improved. These facts led the author to consider the more recent attacks as recurrences of **rheumatism, and not as pyemia.** A recurrence of other infectious diseases under the influence of heterologous infections is not uncommon, and Schnitzler believes that proper observation will show that this is not very uncommon in the case of rheumatism.

M. Behrend[2] discusses **rheumatic exanthems,** and describes a case of erythema exudativum multiforma which involved the throat and larynx, while at first the skin was but little affected. The patient had mitral valvular disease, and there was a rheumatic family history. As this skin eruption is frequently associated with rheumatism, the author believes that the attack was probably rheumatic.

POLYMYOSITIS.

Bacialli[3] contributes a study of polymyositis, with a discussion of the literature. The case which he reports occurred in a man of 39, and began with rheumatic pains, particularly in the upper part of the trunk and in the arms. The movements were limited; the arms swelled, as did the neck; and swallowing became difficult. A physician suspected syphilis, but specific treatment was useless; the swelling advanced over the thorax and lower extremities. Fever was absent. Another physician considered the case acute polymyositis, and ordered theobromin and strychnin. The pains persisted, but the swelling disappeared, although it returned. The patient had been ill 9 weeks when admitted. At that time the limbs, scalp, neck, extremities, and back were much swollen, and there was a papular eruption. Deep pressure was painful. The urine was normal, as were the digestive organs. The condition became worse about every 2 weeks, the temperature rising without any chill. These elevations of temperature persisted for about 2 or 3 days. The examination of the **blood was practically negative, and cultures were sterile.** A portion of muscle was excised during an attack of fever; and from this, and from the blood at that time, the author obtained Staphylococcus pyogenes albus. The patient died after 5 months' illness, with a sudden onset of symptoms of bulbar asphyxia and paralysis of the muscles of respiration. The muscular tissue showed widespread changes, both in the nuclei and in the protoplasm. The nuclei were much increased in number, and often collected in masses. The contractile substance had lost its normal structure, and looked homogeneous and frequently transparent, and of a hyaline or wax-like appearance. The condition is considered by Bacialli to be undoubtedly a **chronic septicemia,** the point of entrance of infection being doubtful, but very possibly the throat. The comparatively benign course he attributes to infection with Staphylococcus pyogenes albus. The organism in this case was scarcely at all pathogenic to animals. The lesions in the muscles were probably produced by the toxin of this organism, through its exertion of an elective affinity for the muscle-tissue.

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[1] Wien. klin. Rundschau, No. 41, 1901. [2] Münch. med. Woch., July 9, 1901.
[3] Il Policlinico, Jan., 1902.

INFLUENZA.

Süsswein,[1] of 21 cases of measles investigated, found bacteriologically that influenza was present in 10. In living subjects he secured cultures from deep within the nasal cavity; in cadavers he investigated the contents of the bronchi, or pneumonic or pleuritic exudates. These results show that **influenza is a frequent complication of measles,** and that the latter is unfavorably influenced by the former; although there are cases in which the influenzal infection may be present, without the clinical evidence of any complication.

Roustan[2] reports a somewhat **noteworthy epidemic which he considered to be one of grip,** which occurred in Cannes and the surrounding region. In a population of 30,000 there occurred as many as 400 cases within a few days; and the epidemic was of such severity that practically all strangers left the town and an actual panic arose. It was thought by many to be an epidemic of typhoid fever. The cases had usually an abrupt onset, with severe headache, rapidly rising temperature, disturbance of the stomach, marked prostration, a course of only a few days, a rapid descent of the temperature, and frequent relapses of short duration. The attacks were considered to be comparable to nothing but influenza. [There was apparently no bacteriologic study of the cases.] None of the patients showed rose-spots, bronchitis, or diarrhea. The Widal reaction was regularly negative. There were very few cases of typhoid fever in Cannes at the time, and the number of such cases had been decreasing in the town. The severity of the epidemic was shown by the fact that 21 persons died. No autopsies were carried out.

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[1] Wien. klin. Woch., No. 47, 1901

[2] Bull. de l'Acad. de Méd., Dec. 3, 1901.

Neumayer[1] discusses **pseudocavity signs in influenzal pneumonia.** Signs of cavity, such as pseudocavernous breathing, pectoriloquy, etc., occasionally occur in ordinary lobar pneumonia, in metapneumonic abscess, in enlargement of the peribronchial glands, and in some other conditions; but it is almost always comparatively easy to determine that tuberculous cavities are absent, except in grippal pneumonia. The important signs distinguishing these two conditions are that the signs of cavity develop slowly in tuberculosis, even when the disease is running a rapid course; while in influenzal pneumonia the signs of cavity appear in 2 or 3 days. The signs of real tuberculous cavity persist and tend to increase in size, very rarely showing a tendency to disappear; while the pseudocavity signs are very variable, and often disappear after a few days, although sometimes they may be prolonged over a considerable period. The general character of the patient and the general symptoms are often of the most extreme diagnostic importance, although it is to be remembered that the convalescence from influenzal pneumonia may strongly suggest tuberculosis. Fever, however, is likely to be absent in this condition, and present in tuberculosis. There is little expectoration in the convalescence from influenza. In general, the onset of the disease, its course, the pneumonic fever, and the recognition of a pneumonic process, make the diagnosis comparatively clear.

C. Moore,[2] in discussing the **ocular complications of influenza,** directs attention to the sensitiveness of the ocular apparatus to the influenza toxin. Ocular affections in influenza are, however, usually of good prognostic significance, unless, as occasionally happens, they are the result of metastatic or embolic processes.

Ophüls[3] reports the case of a man who was taken ill with chill, cough, and general prostration. He had fever, some slight jaundice, distention of the abdomen, and enlargement of the spleen and liver; the lungs were normal. The patient died after about a week, with increasing icterus and higher fever. The mucous membrane of the rectum was found inflamed and ulcerated, with numerous small abscesses in the submucosa; there were also numerous abscesses in the liver, a septic thrombosis of the hepatic vein, and septic infarcts in the lung. The abscesses in the rectum and liver and the contents of the gall-bladder yielded, upon culture, some streptococci; but also large numbers of a peculiar **bacillus, which closely resembled the influenza bacillus,** although it showed some variations upon culture. Injection into animals was negative. The case was evidently one of **infection of the rectum,** the other conditions being secondary. The patient had apparently never had pneumonia, and the definite way in which infection had occurred was obscure.

PNEUMOCOCCUS INFECTION.

A. Prochaska[4] briefly reports 4 extremely interesting cases, which were apparently instances of **general pneumococcic sepsis without**

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[1] Gaz. des hôp., 1901, No. 98.

[2] Albany Med. Ann., 1901, No. 7.

[3] Am. Jour. Med Sci., Dec., 1901.

[4] Deut. med. Woch., May 22, 1902.

pneumonia. He refers to his very regular observation of the presence of pneumococci in the blood of pneumonia, and describes the four cases above mentioned, in all of which there were severe symptoms associated with bronchitis and with the presence of pneumococci in the sputum and also in the circulating blood. In none of the cases was there pneumonia clinically; and one case died, and showed postmortem the absence of pneumonia. In the first case there was a **hemorrhagic nephritis, which followed an acute bronchitis.** The urine in this case contained pneumococci. In the second and third cases there was slight bronchitis, with high fever and severe chill, followed by recession of the symptoms and disappearance of the bronchitic signs; but afterward there were again marked chills, without any physical signs, but with the presence of pneumococci in the blood. Malarial plasmodia were absent. The spleen was palpable. The fourth case had had bronchitis for 3 weeks and was improving. There was suddenly an attack of fever, with cyanosis and collapse. The blood in this case was so loaded with pneumococci that they could be easily discovered in ordinary smear preparations. This patient died; and no lung changes were found except hypostasis. The author thinks that these cases were **probably infected through the bronchi.** There was no evidence of infection through the tonsils.

Raw[1] insists upon the importance of the pneumococcus in causing lesions elsewhere than in the lungs, and describes the occurrence of **7 cases of pneumococcus arthritis in 817 cases of pneumonia;** *i. e.*, nearly 1 %. Of the 7 patients, 3 died. In all the cases the disease, both of the lungs and of the joints, was on the right side. The knee was affected three times; the shoulder-joint, twice; and the ankle, once. In two instances the exudate was serous; in the others, purulent. The joint trouble was repeatedly noticed before the pneumonic signs, and it was often noticed that slight signs of arthritis disappeared directly after the crisis. Pneumococcic arthritis seems to be more common and more severe in alcoholics than in other persons. The only rational treatment is draining the joint.

C. Cary and J. P. Lyoh[2] describe a case of **pseudomembranous inflammation of the mucous membranes of the lips, mouth, and throat,** the conjunctiva, the prepuce, and the anus, caused by the pneumococcus. These organisms were found in large numbers, to some extent mixed with staphylococci. The diphtheria bacillus was absent. The involvement of the pseudomembranes followed a double pneumonia. The patient recovered, the fever disappearing by lysis.

MICROCOCCUS CATARRHALIS INFECTION.

A. Ghon, H. Pfeiffer, and H. Sederl[3] contribute an extensive and valuable discussion of Micrococcus catarrhalis as a **cause of disease.** Gohn and Pfeiffer have made careful bacteriologic **studies of the sputum in 140 cases,** and, whenever the opportunity occurred, have made postmortem examination of these cases. They give extensive descriptions of the cultural and morphologic characteristics of the organism, and state that they consider Micrococcus catarrhalis, Micrococcus gonorrhœæ, and Micrococcus meningitidis to constitute a group of closely related micro-organisms, which, however, show definite individual characteristics. The **diagnosis of Micrococcus catarrhalis is, they state, not difficult.** It exhibits the general type of the gonococcus, although it is slightly larger than the latter, decolorizes more quickly and more homogeneously by Gram; and it exhibits homogeneous, coarsely granulated colonies, of a yellowish-brown color, with irregular edges that look as if they had been nibbled. There is a weaker formation on the surface of bouillon cultures; in stab cultures the organism grows only superficially. The growth in later generations increases in activity. The organism has only slight pathogenicity for the animals usually investigated. As to its pathogenic importance for man, the authors found it absent in 31 % of the cases; hence, it is not a regular inhabitant of the air-passages. In the 81 cases in which it was present it was found in largely varying numbers. **In 1 case it was the only organism present.** This was that of an infant that died with a purulent bronchitis. They believe that in this case the organism must be considered as the cause of the disease. In 5 other cases it was present in such very large numbers as compared with other organisms, and showed so close a relation to the course of the disease, that they believe that it undoubtedly produced the condition. In 6 other cases it probably caused the condition, or, at least, was the chief factor in it. In 51 cases it was present in such comparatively small numbers that it is likely that it had but slight, if any, importance as a cause of the condition. Sederl discusses the 5 cases which have been referred to as having probably been **due chiefly or entirely to Micrococcus catarrhalis.** They closely **resembled either influenza or lobar pneumonia,** and their diagnosis from these conditions was made only by means of the bacteriologic studies. He decides that Micrococcus catarrhalis may be considered to be the cause of the disease, and that it can, of itself, produce simple bronchitis, lobular pneumonia, or a clinical picture which corresponds with lobar pneumonia. It produces not only local symptoms, but also more or less severe general ones. The conditions caused by it, however, show in themselves nothing characteristic; they very closely resemble the diseases caused by the influenza bacillus and the pneumococcus, and it is very probable that they are frequently found with such conditions. Micrococcus catarrhalis is also found in mixed infections of the respiratory tract; and probably, also, in those cases they at times play the primary rôle in producing the disease. In other cases it is likely that the micrococcus acts only as a saprophyte.

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[1] Brit. Med. Jour., Dec. 21, 1901. [2] Am. Jour. Med. Sci., Sept., 1901.
[3] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 3 u. 4.

CEREBROSPINAL FEVER.

Albrecht and Ghon[1] report 22 cases of **epidemic cerebrospinal meningitis** in which they determined the presence of Diplococcus intracellularis meningitidis. They prefer to call it Micrococcus meningitidis cerebrospinalis, as indicating the nature of the organism and its etiologic importance. They consider that it is undoubtedly the cause of the one form of cerebrospinal meningitis, in most instances; and that this form may appear both sporadically and epidemically.

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[1] Wien. klin. Woch., No. 41, 1901.

Nuttall and Hunter[1] report that in 10 cases of cerebrospinal meningitis, by carrying out lumbar puncture, they **isolated a diplococcus** which had the morphologic and biologic characteristics of Diplococcus intracellularis meningitidis. It was observed in 2 forms, which were very different; sometimes in pure culture and sometimes mixed with other microorganisms. The clinical picture of the cases which they observed, and the pathologic changes, were those of **posterior basal meningitis,** which disease they consider to be probably a **sporadic form of cerebrospinal meningitis,** and to be due to the same microorganism.

W. J. Buchanan[2] states that in 4 years he observed, **in Bhagalpur, Bengal, 60 cases of cerebrospinal meningitis,** 43 of which died. Of the fatal cases, 22 were foudroyant; 17 ended between the third and the fourteenth day; 4 were more chronic, lasting from 2 to 3 weeks. Three cases were particularly interesting. One followed a subacute polyarthritis; one, in a man of 55 years, began with a right-sided hemiplegia and aphasia, and showed on postmortem, as an explanation, an unusually thick and dense purulent exudate in the left hemisphere; the third, which died 3½ hours after the onset of the disease, showed severe congestion of the dura, a beginning leptomeningitis, and hemorrhagic infiltration of the surface of the brain. The nature of these cases was definitely determined by positive cultures of Diplococcus intracellularis.

R. Tschinkel[3] reports a case of **purulent meningitis which complicated a chronic catarrhal rhinitis** and ended in 32 hours. Cultures of blood from the finger and from the vein were negative, but **numerous meningococci** were obtained from the cerebrospinal fluid, which was gotten by lumbar puncture.

E. Bendix[4] discusses the **diagnosis of meningitis by means of the examination of the cells in the exudate.** He reports 8 cases of meningitis, 5 of them of tuberculous origin and 3 sporadic cases of epidemic cerebrospinal meningitis. The diagnosis was confirmed in all cases by autopsy. The cases will be reported in detail elsewhere; the main point of the present report is that in the 5 tuberculous cases the specific organisms could be found in but one instance, while in all 5 cases it was found that the cells in the exudate were almost entirely lymphocytes. In one of the 3 cases of meningococcus meningitis he found a majority of lymphocytes. This, however, was a case which ran a slow course, and Bendix decided that the **presence of a majority of lymphocytes in the exudate means merely that the case is running a slow course,** and does not mean that it is due to any special infection. This is in consonance with the statements of Ribbert that the cellular exudate in inflammations is composed chiefly of polymorphonuclear leukocytes when the inflammation is acute, and of lymphocytes when the inflammation is of slow course.

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[1] Brit. Med. Jour., Sept. 21, 1901.

[2] Lancet, July 13, 1901.

[3] Prag. med. Woch., No. 46, 1901.

[4] Deut. med. Woch., Oct. 24, 1901.

ERYSIPELAS.

Jordan,[1] in a discussion of the etiology of erysipelas and its relation to pyogenic infections, reaches the conclusion that erysipelas is etiologically **not a specific disease;** that typical erysipelas can be produced in the rabbit's ear, not only with streptococci, but also with staphylococci, pneumococci, and colon bacteria; that human erysipelas is usually caused by Streptococcus pyogenes, but can be produced by Staphylococcus aureus. The question whether the facultative pus-producing organisms, such as the pneumococcus, the colon bacterium, and the typhoid bacillus, can produce erysipelas in human beings is still unsettled. These observations the author considers to be entirely in accord with clinical conditions. He believes that it is impossible to separate strictly, from a clinical standpoint, erysipelas and pseudoerysipelas, as these vary only in their intensity.

GONOCOCCUS INFECTION.

Ware[2] reports the case of a patient who had acquired gonorrhea 2 months earlier, and who had meanwhile had pain in the knee-joint. He was suddenly attacked with **pain in the muscles of the shoulder,** the shoulder-joint itself apparently being uninvolved. The painful muscles were swollen. Pus was thought to be present, and an incision was made. No suppuration was discovered, but the muscles were found to be infiltrated with a serous fluid. Smear preparations were made from this fluid, and micro-organisms having the morphology of gonococci were found. The patient then proceeded to entire recovery.

ACTINOMYCOSIS INFECTION.

Van der Veer and Elting[3] give a résumé of the subject of actinomycosis, with the report of a case of **abdominal actinomycosis.** The patient was a carpenter who had suddenly experienced abdominal pain, which gradually disappeared. Afterward the patient noticed swelling of the abdomen. A hard infiltration was found in the region of the umbilicus, and a mass which was thought to be the omentum was present. There was slight leukocytosis. The tumor was removed by laparotomy, without being certain of its nature, though it was thought to be a sarcoma. Later, an abscess was opened in the discolored skin surrounding the umbilicus, and typical actinomycetes-pus was emptied. The patient was then given large doses of potassium iodid; he gradually improved, the discharge stopped, the infiltration disappeared, and the man **recovered after months of treatment.** During the course of his disease he had, **for a time, a secondary autoinfection on the side of the nose.**

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[1] Münch. med. Woch., No. 35, 1901.

[2] Am. Jour. Med. Sci., July, 1901.

[3] Albany Med. Ann., Jan., 1902.

ANTHRAX INFECTION.

Kreissl[1] reports the case of a man who was admitted into the hospital in an unconscious condition, and who soon died. The postmortem showed an exudate in the right pleura, infiltration of the intraperitoneal and subperitoneal lymph-vessels and lungs, bronchopneumonic areas in the right upper lobe, edema of the mediastinal tissue, and bloody infiltration of the glands. The pia mater also showed widespread hemorrhages; the cortex of the brain was edematous. The **lungs and the meningeal exudate were found to contain large numbers of anthrax bacilli.** The skin, gastrointestinal tract, and upper air-passages were free. The **source of the infection could not be determined.**

STREPTOTHRICAL INFECTION.

J. H. Musser[2] discusses streptothrical infection and **reports 2 cases.** The first patient, a man of 37, showed increased respiration and pulse and rise of temperature, with signs of bronchitis; and had mental disorder. There was an increasing degree of cyanosis. He finally died with the signs of bronchopneumonia. There was no leukocytosis; indeed, the white corpuscle count was 3200. The study of the sputum showed the presence of the streptothrical filaments. There were also numerous streptococci present. The case was either one of primary streptothrical infection, or one of primary streptococcus infection with a secondary streptothrical growth; although it is possible that the organisms described were branched forms of the tubercle bacillus. The second patient had **sudden epileptiform attacks,** with right-sided weakness; and also had a sudden collapse, with semiconsciousness, exaggeration of the reflexes, the presence of the Babinski phenomenon on the right side, positive Kernig's sign, and ankle clonus. There was decided edema of the optic disks. Lumbar puncture caused some improvement; but the patient grew worse again, and died a week after the first convulsion. Autopsy showed an **abscess posterior to the fissure of Rolando,** containing chocolate-colored and grayish-yellow, oily fluid, the pus being very foul. Microscopically, there were no signs of inflammation over the abscess, and no evidence of vascular disease; and there was no bone necrosis or ear disease. There was no tuberculosis; but, after a long search, streptothricæ were found. Cultures were negative; guinea-pigs were inoculated, with negative results. Musser states that the streptothrix may be pathogenic to man, though its presence may be merely secondary to another infection; and that, in pulmonary cases especially, death is due to a mixed infection. The morphologic relation to tubercle bacilli and allied organisms should be remembered.

DENGUE.

H. Graham[3] describes an epidemic of dengue in Beyrout, Syria, and that neighborhood, which was widespread and severe. He believes that he has **demonstrated with the greatest probability that the disease was carried by the mosquito.** He mentions a number of cases in which persons in the same house with patients were protected from mosquitos, and escaped infection; while, when these precautions were not carried out, infection usually occurred. He also states that he has conveyed the infection by allowing Culex to bite patients with dengue, and afterward other persons who had not had the disease. He particularly notes that mosquitos that had bitten dengue patients were carried to the mountains, where the disease had not occurred at all; and that after they had bitten young men who had not been out of that region, dengue appeared in these persons, the disease occurring from 4 to 6 days after the patients had been bitten by the insects. The author also states that he has **observed an ameboid object in the blood of patients with dengue.** This ameba resembles Plasmodium malariæ, but has a much longer cycle of life. Its development was, therefore, much more difficult to follow. The fresh blood was the most satisfactory for observing this parasite. This hematozoon is unpigmented. Graham states that it is constantly present during the fever. He believes that Culex carries the disease.

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[1] Wien. klin. Woch., No. 42, 1901. [2] Chicago Med. Recorder, Aug., 1901.
[3] Med. Rec., Feb. 8, 1902.

BUBONIC PLAGUE.

W. Kolle and E. Martini,[1] in a discussion of plague, draw attention to the importance of the cutaneous method of experimental infection devised by Weichselbaum, Albrecht, and Ghon. They give examples of the fact that by this method it is easy to demonstrate various degrees of virulence in plague bacilli. Mice and rabbits are very poorly fitted for tests of the virulence of the plague organisms. For increasing the virulence of these organisms, they find an **inhalation apparatus invented by Martini** to be of importance. By means of this, pneumonia can readily be produced in rats, without danger to the operator, and the injection of the juice of their lungs into other animals produces a growth of plague bacilli of very high virulence. The authors call especial attention to the importance of their observation that **rats may suffer from chronic plague.** This fact may be a very clear explanation of a good many instances of prolongation of plague epidemics, or of the appearance of cases of plague after opportunity for infection has apparently disappeared. In making cultures of plague bacilli, the authors insist upon the importance of using small amounts of blood—from 1 to 20 drops—in about 50 cc. of bouillon, whereby the bactericidal effect of the extravascular blood is overcome.

Corthorn[2] has investigated a large series of cases of plague for the **presence of albuminuria,** making daily examinations. It was found to be **absent in only 14 %** of the specimens. Its amount varied greatly, and was usually more marked in the fatal cases than in those that recovered. In the latter, albuminuria always appeared as early as the fourth day. In by far the larger number of cases it lasted about a week; in a good many, as long as 2 weeks; and in some, from 2 to 5 weeks. Hyaline and granular casts were frequently found. **Plague bacilli were never found in the urine,** even by means of cultures. Blood was present in a number of instances; in 3, in large amounts. Retention of urine was seen in 2 % of the cases that ended in cure, and in 12 % of the fatal cases.

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[1] Deut. med. Woch., Jan. 2, 1902.

[2] Brit. Med. Jour., Sept. 14, 1901

Treatment: Prophylaxis.—W. Kolle and E. Martini[1] insist emphatically upon the **importance of rats and mice in carrying the disease.** They think, however, that **the relation of fleas to the transmission of the disease has been much overestimated.** There should be strict measures adopted to destroy rats, as far as possible; and, particularly, there should be international regulations concerning the destruction of these animals on board ships. The authors think that if the rodents on shipboard could be absolutely exterminated for a few years, plague would be driven back to the regions in which it is endemic. They also emphasize the fact that bacteriologic studies of epidemics among rats and mice are of the utmost importance, and should be regularly carried out when there is any possibility of the occurrence of plague. They have little faith in the use of protective inoculations; partly because of their inefficacy, largely because of the impossibility of carrying them out with large numbers of people. The use of plague serum as a curative measure, while not very encouraging, has indicated that it is at least worth while to carry the study of this question further.

The same authors[2] have investigated the **influence of plague serum in rats, mice, and guinea-pigs.** They decide that when very virulent cultures are used, whatever the process of infection, the serum seems to be useless, so far as preserving the lives of the animals is concerned. Further, with cultures of weak or moderate virulence, no noteworthy effect could be obtained if the injection of serum was given as long as 24 hours after infection, or at a time when the animals already seemed ill. It was, however, notable that in almost all the animal investigations the serum **seemed to prolong life somewhat.**

The Report of the Municipal Commissioner on Plague in Bombay (1901) states that the mortality in 1899–1900 was 71,801, being 97.02 per 1000. From August 31, 1899, to May 30, 1900, **163,973 inoculations with Haffkine's prophylactic** were carried out. The writer of the report gives a number of striking instances of the **apparent protective influence of the inoculations.** In one house, in which there were 61 persons, 24 were inoculated; and 20 cases of plague afterward occurred in the house, only 1 being in the inoculated. This person recovered, while 12 of the uninoculated died. Other instances were not so favorable. There are a number of reports on Lustig's serum, that of Mayr showing 403 cases treated with the serum; 154 recovered, the rate of recovery being 38.2 %; while with the usual treatment the rate of recovery was only 19.5 %. Another report, by Polverini, shows a recovery rate of 39.36 % in those treated with the serum, while in those treated otherwise the recovery rate was 19 % to 21.3 %. A third report, by Childe, shows a reduction of the mortality from 80 % down to about 69 %.

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[1] Deut. med Woch., Jan. 23, 1902.

[2] Deut. med. Woch., Jan. 9, 1902

W. B. Bannerman,[1] in writing of **inoculation in the incubation stage of plague,** mentions the view that inoculation with Haffkine's vaccine in the incubation period would probably aggravate the disease considerably. He reports a large series of cases, observed by different men, which he believes to refute the above view entirely. The mortality in those inoculated in the early stages of plague was only about 48 %, while the general mortality was considered to be between 75 % and 85 %. He believes it to be **desirable to inoculate at once,** if a person has been exposed to infection; and thinks that the cases which he presents also show that Haffkine was right in stating that the prophylactic begins to act with benefit only after a lapse of 24 hours.

Lignières[2] states that in the treatment of plague at the Lazaretto at Rosario in 1899–1900, about **90 % of recoveries** were obtained in the cases in which the serum was used early. He also found that the intravenous injection of plague serum was well borne, if carefully carried out. He recommends the use of 60 cc. for the first dose, and 40 cc. within from 12 to 24 hours.

RABIES.

Kraus and Clairmont[3] note that previous investigations have indicated that birds, especially pigeons, while susceptible to intoxication with rabies, usually recover spontaneously. The authors have **injected rabies toxin into a large series of birds,** and have, with few exceptions, **produced death by subdural injection.** They found changes in the central nervous system analogous to those found in man and the higher animals. Ravens and old pigeons were refractory. The authors found, however, that neither the blood-serum nor the brain-substance of these refractory birds destroyed the poison.

Kraus[4] presents some investigations which he believes demonstrate that the **bile has a distinctly destructive effect upon the rabies virus.**

Dadi[5] investigated the central nervous system of a large series of animals that either had or were suspected of having rabies; and also specimens from 4 patients who died of that disease. He examined the vagus nucleus and usually portions of the cerebrum, cerebellum, and spinal cord. In each case in which rabies was present, he found round-cell infiltration, hyperemia, and the chromatolysis and vacuoles in the ganglion cells that have been described by others. The changes described by Nelis and van Gehuchten he found only in those animals that became diseased by natural methods, not in those that were artificially infected. He **does not believe that it is possible to make an absolute diagnosis through the simple examination of the nervous system,** for the characteristic changes may be absent. If present, however, they are of the utmost importance in diagnosis.

[1] Brit. Med. Jour., Sept. 14, 1901.
[2] Ann. de l'Inst. Pasteur, Oct., 1901.
[3] Zeitschr. f. Hyg. u. Infektionskrank., Bd. xxxiv.
[4] Zeitschr. f. Hyg. u. Infektionskrank., Bd. xxxiv.
[5] Rivista critica di clin. med., 1901, No. 26.

LEPRA.

L. Glück[1] discusses the **initial lesion of leprosy.** It has not been definitely established that there is an initial lesion similar to that which occurs in syphilis. After studying 200 cases, the author decided that in all there were signs of an initial lesion for a considerable period before other evidences of the disease appeared. On the basis of these observations and others recorded, he asserts that there is an initial lesion, and describes it as a round, well-limited, flat, slightly elevated, infiltrated area, of coppery-red color or sometimes darker, which grows paler in the middle and becomes depressed, but may spread to the size of the palms of the hand. Later on, it becomes very pale and atrophic, shows little or no scaling, becomes more or less anesthetic, and is surrounded by a wall of infiltrated tissue. The **initial lesion produces no subjective symptoms,** and, for this reason, is **frequently overlooked** by the patient and is not shown to the physician. For months or years it may be the only evidence of leprosy. It is **most commonly found on the extremities;** more rarely, on the face; very rarely, on the trunk. It may appear on the mucous membranes, particularly that of the nose; but although the author thinks that he has observed one definite case of primary lesion of the mucous membrane of the nose, he insists that this is not its ordinary situation. He believes that leprosy has been transmitted through vaccination.

Strain[2] reports a case of **leprous infection through a wound.** A woman of 54 who lived with her leprous son was operated on for carcinoma of the breast. The wound healed during ambulatory treatment. About 6 months after the operation, small, hard nodules, surrounded by a red area, appeared at the sternal end of the wound. Two weeks later red spots appeared on the face, and the skin became infiltrated; the arms were involved, and showed some anesthesia. There were also various anesthetic areas on other portions of the body. The diagnosis of leprosy was considered to be definitely established, and the infection was thought undoubtedly to have occurred through the wound.

TUBERCULOSIS.

Etiology and Pathogenesis.—Baumgarten[3] states that while it has previously been possible to produce only miliary tuberculosis of the lungs, he **has been able, by injecting virulent tubercle bacilli into the normal urethra or urinary bladder, to cause typical phthisis of the upper lobes of the lungs in animals.** The gradual entrance of the bacilli into the body, which occurs in this way, gives these organisms an opportunity to seek their location of preference in the lung apices. It is customary to consider the involvement of the apices of the lungs as absolute evidence of the truth of the inhalation theory; but Baumgarten's experiments make it seem possible that the ordinary **pulmonary**

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[1] Wien. med. Woch., 1901, Nos. 29–31.

[2] Brit. Med. Jour., Sept. 14, 1901.

[3] Wien. med. Woch., No. 44, 1901.

tuberculosis of human subjects may have a hematogenous origin, as has been suggested by some other authors.

H. Ribbert[1] contributes an interesting discussion of a previous publication of his own, which appeared in an obscure report, and has, therefore, not come to the notice of the profession. He insists that **in ordinary cases of phthisis a hematogenous infection must be reckoned with;** for in miliary tuberculosis he finds that the tubercles are largest and most advanced in the apices, and decrease from here downward, in number and size. The nodules are also found in the two lungs in different stages of advancement. It is only necessary, therefore, to consider that a patient may be fairly resistant to tubercle bacilli in order to realize that hematogenous tuberculosis may produce conditions entirely similar to those seen in ordinary phthisis. The portions of the lung-tissues involved in ordinary hematogenous tuberculosis are exactly the same as those involved in the condition usually considered inspiration tuberculosis. The bacilli do not set up disease immediately about the vessel-walls, but pass into the tissues and involve the smaller bronchioles or the outlets of the alveoli. There is, therefore, no anatomic difference between inspiration tuberculosis and hematogenous tuberculosis. The author believes that the most common infection is through the lungs, but that this may often be without primary involvement of the lung-tissue itself, the **bacilli being arrested in the bronchial glands, and from here causing involvement of the lungs.**

St. Clair Thomson,[2] in a discussion of tuberculous infections through the air-passages, reaches the conclusion that the **most common mode of tuberculous infection is by inhalation,** and that the inhaled bacilli **infect the system before the air-current reaches the larynx,** and not ordinarily through direct infection of the pulmonary tissue. The infection most probably occurs through the lymphoid tissue of the nasopharynx and pharynx. The author considers that the common view that the bacillus is inhaled directly into the pulmonary alveoli is unjustified.

P. Grawitz[3] contributes a paper on the **point of entrance of the tubercle bacilli** and the localization of these bacilli in human beings. From a consideration of the fact that intestinal tuberculosis is comparatively rare, and that in a great majority of the cases of intestinal tuberculosis we must believe that the infection was due to the swallowing of tuberculous sputum, one must reach the conclusion that those cases in which there is probability of a primary tuberculosis due to infection through the food are very rare. But one cannot decide offhand in this way that infection through food is an extreme rarity. Grawitz himself, in the last 1104 necropsies which he has carried out, found 18.66 % of active tuberculous processes, and 8.68 % of cured tuberculosis, and there were **only 4 instances** in this whole series **in which there was tuberculosis of the intestine or mesenteric glands without tuberculosis of the lungs** from which the intestinal infection might have occurred. He believes that the intestinal mucous membrane is comparatively resistant to the entrance of tubercle bacilli. He has been constantly observing the tonsils with more care, however, and has in many instances found giant-cells and bacilli even when the tonsils were small and macroscopically seemed small. From the tonsils the bacilli may easily reach the lymph-stream and cervical glands, and from here may infect the lungs, pleura, and pericardium, and the patient may die without the faintest evidence of tuberculosis of the mesenteric glands or intestines, and consequently without the evidence that is usually demanded that the infection was from the food. It is extremely difficult, in most cases of this kind, to demonstrate positively that the infection was from the food. In order to do that, one should not confine one's attention to postmortem examinations, but should make careful clinical observations concerning the way in which the disease appeared in the various organs, and should also make microscopic examinations of the tonsils in a series of cases of chronic enlargement of the cervical glands, probably of tuberculous nature. Nevertheless with our present knowledge it is practically certain that a great **many cases of tuberculosis are due to infection through the food,** and this may in many cases be due to primary infection through the tonsils.

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[1] Deut. med. Woch., April 24, 1902.

[2] Practitioner, July, 1901.

[3] Deut. med. Woch., Oct. 10, 1901.

V. Körosy[1] has made a study of the **mortality of children from tuberculosis in relation to the age of the parents,** and finds that when the mother was less than 20 years old, the mortality was 8.45 in 100, decreasing from this point down to 2.78 when the mother was between 30 and 35; and rising slightly after this age. A similar condition of affairs was observed in relation to the father's age. If he was below 25, the mortality was 5.71, decreasing from this point up to the age of 40, and after that rising again.

H. M. King,[2] in studying the **relation of heredity to immunity and to selective activity in tuberculosis,** has investigated 245 consecutive cases. He finds that 1 in 4 gives a history of phthisis in the parents, and 1 in 3 a history of the disease in a brother, a sister, or both. More than two-thirds of the latter have no phthisical parents. A much longer period was found between the supposed exposure to infection and the subsequent appearance of the disease in those who had a phthisical parentage than in those who had not. Of 103 fatal cases, the average length of life after the development of the disease in those having a history of phthisis in the parents was as 4 to 3. The author concludes that the percentage of consumptives having a tuberculous parentage is smaller than that of those having a nontuberculous one, and decidedly smaller than is accounted for by the additional risk of infection to which the former class is subjected. He believes that **tuberculosis in the parents confers upon the offspring a relative immunity,** which is not sufficiently protective to prevent the incidence of the disease, but is still demonstrable.

C. A. Blume[3] reports an extremely careful study of 486 persons with tuberculosis. He particularly discusses the question of the **spread of tuberculosis in families,** and reports that he could determine infection from the parents in 85 cases, marital infection in 23, and infection from other persons in the household in 13. He considers these figures to be fair illustrations of the frequency of infection from different sources in the family, and insists that infection from individuals within the household is extremely important. He believes the infection usually comes from the living subject of tuberculosis, and that infection through dried sputum is less important. Isolation he considers of extreme importance, so far as it can be carried out. Sanatoriums are of much importance in the control of tuberculosis, but they have little effect as compared with the influence that proper regulations concerning prophylaxis may exert.

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[1] Rév. de la tuberc., Aug., 1901, p. 282.

[2] Med. Rec., Oct. 12, 1901.

[3] Hospitalstidende, Nos. 44 to 51, 1901

M. Fishberg[1] considers that the Jews have less predisposition to tuberculosis than the general mass of the population, and presents statistics to substantiate this view.

H. Coates[2] has investigated the question of **house-infection in tuberculosis.** He divides the houses examined into the classes: (1) those which were dirty and had had careless tuberculosis inmates; (2) clean houses occupied by careless consumptives; and (3) dirty houses in which there had been no cases of tuberculosis for years. Of the first class, 21 showed 14 instances of positive tuberculous infection of the house. In 8 of these 14 instances the lighting and ventilation were bad; in the 7 negative instances the lighting and ventilation were good. Ten houses that were clean, but infected, showed infectious dust in 5 instances. The average cubic air-space per individual was much lower in these houses than in the unaffected five, and the light and ventilation were poor. The 10 dirty houses without consumptive inmates showed negative results in all instances. The waiting-room of the Out-patient Department of the Manchester Hospital for consumptives—a room daily visited by about 180 patients—was tested with negative results. This room was well lighted and ventilated, and had a high ceiling. These results show the importance of light and air as disinfectants for tuberculosis.

H. W. Buckler,[3] in a discussion of **pulmonary tuberculosis in Baltimore,** states that in 25 years there have been 28,479 deaths from phthisis—a ratio of 12.8 % to the total mortality. In the last five years of the investigation the percentage mortality had fallen from 11 to 9.6. The percentage mortality was lower in Baltimore than in any other of 6 prominent cities of this country, in spite of the large negro population. In one region of the city, which, in spite of being thickly settled, showed very few deaths from phthisis, the author believes that the low tuberculosis rate was due to the presence of large gas-works, which impregnated the air with fumes from the furnaces and rendered the atmosphere to some extent germicidal. Tuberculosis is evidently a disease of filth and bad hygiene, and various regions of Baltimore show this most strikingly. The bad hygienic conditions under which the negroes live are chiefly responsible for the marked morbidity among them. Buckler notes one family in which 3 deaths from tuberculosis had occurred within 2 years, and it was found that the previous occupant of the house in which the family were living had died of lung trouble shortly before they took the house. Certain regions seem to be more especially affected than their hygienic conditions would lead one to expect.

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[1] Amer. Med., Nov. 2, 1901. [2] Med. Press and Circ., Aug. 7, 1901.
[3] Johns Hopkins Hosp. Bulletin, Sept., 1901.

Lannelongue, Achard, and Gaillard[1] state that, from their experiments, they have decided that moderate cold, or, indeed, **moderate changes in temperature** of any kind, **have little influence upon the development of experimental tuberculosis;** but sudden reductions of temperature, which have no noteworthy influence upon healthy guinea-pigs, cause tuberculosis to run a rapid course.

A. Maffucci[2] reports an experimental investigation concerning the **toxin of tubercle bacilli and the infectiousness of the semen of tuberculous animals.** He finds that both the toxin and the bacillus may be found in the semen of infected animals; and that apparently the toxin may make the female sterile, or the bacilli may produce infection. There is a marked tendency for females to abort after being impregnated by tuberculous males, and the young of tuberculous males are likely to die early without tuberculous lesions. The females impregnated by tuberculous males are likely to die of marasmus, whether they have aborted or have gone to full term. The injections of tuberculin into the vagina produced a similar condition of marasmus; and the females in general, as a result of impregnation with tuberculous males, showed a greater tendency to a condition of general intoxication than to specific tuberculosis.

J. A. Grober[3] reports some studies of pleurisy. He first refers to **1000 cases of phthisis,** of which he investigated the histories for a record of pleurisy, and found that **in 88 persons the previous history showed an attack of pleurisy.** Sixty-eight persons knew that they had had pleurisy during the course of the phthisis. The percentage first mentioned (8.8) is high, considering the great frequency of tuberculosis of the lungs. The author believes that it adds emphasis to Penzoldt's statement that **every person with pleurisy should be suspected of phthisis.** Of the 1000 cases of the latter disease, 400 showed a family tendency to it. The great majority of the cases were seen between the tenth and fortieth years of life. As to the etiology of pleuritis, Grober refers to the 200 cases which have been treated in the clinic at Jena in the past few years. Of these, 140 were in males, and 60 in females. According to etiology, they were divided as follows: tuberculous, 21; probably tuberculous, 22; postrheumatic, 7; postpneumonic, 8; postinfluenzal, 2; traumatic, 12; occurring in heart disease, 5; occurring in malignant growths, 5; in other infections, 12; idiopathic, or of unknown cause, 106. Of the last group, 30 showed distinct family tendency to tuberculosis; it was, therefore, thought that they should be properly included in the cases of tuberculous pleurisy. If this were so, the **tuberculous cases would constitute over 36 % of the total.**

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[1] Gaz. hebd. de Méd. et de Chir., No. 85, 1901.

[2] Rivista critica di clin. med., Nos. 7 and 8, 1902.

[3] Centralbl. f. innere Med., Mar. 16, 1902.

Baldwin[1] reports 6 cases of **unusual localization of tuberculosis.** In one case of miliary tuberculosis there was tuberculous involvement of the hypophysis; in another, of the thyroid gland; in the others, there was local tuberculosis of the uvula, of a fallopian tube, of the intercostal tissue next the sternum, and of an adenomatous polyp of the uterine cervix.

Symptomatology.—Naumann[2] considers that tuberculous subjects are by no means always inclined to carry their disease happily. He finds, on the contrary, a very **distinct tendency to depression** and that their **intellectual capability is likely to decrease.** The conditions of life in institutions also reduce their psychic activity, particularly when, as is frequently the case, they are suffering from general nervous depression. The author, therefore, advises that tuberculous subjects be provided with some regular occupation and interests.

C. R. Upson[3] discusses **latent pulmonary tuberculosis.** He reports upon the cases of 197 persons who consulted him for other affections than those of the lungs, and who were yet found to be tuberculous. The personal and family history were good in most instances, and in 99 cases the home environment was also good; 78 persons had lived or worked with consumptives; 48 lived in poor boarding-houses. In a majority of cases there was a slight daily rise of temperature, and in over half the respiratory movements and pulse-rate were somewhat rapid. Twelve patients had spat up blood; in two women this had occurred for several months at the catamenial period.

E. Klebs[4] reports that he has very frequently found Diplococcus semilunaris in tuberculous cases, particularly on the tonsils. He has found the organism, however, in tuberculous disease of the bones and joints, and in tuberculosis of the skin and in other tuberculous processes. The organism is distinguished from the meningococcus and from the gonococcus by its variable size and by its being solely extracellular. Its variations in size distinguish it from the pneumococcus. The author believes that it is of much importance in cases of tuberculosis of the glands, considering that it prepares the tissues for the invasion of the tubercle bacillus through the tonsils. He thinks, therefore, that **scrofulosis is, in most instances, a mixed infection** with the tubercle bacillus and Diplococcus semilunaris. He considers it probable, also, from observations on human subjects and from animal experiments, that it may further the invasion of the meninges with the tubercle bacillus; and he believes that **the diplococcus is the cause of the atrophy** of the blood-building organs—the actual phthisis—which occurs in tuberculosis. In tuberculosis of the bones and joints, he thinks that the disease takes on an acute course only when Diplococcus semilunaris complicates the condition and begins to develop actively.

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[1] Med. News, Dec. 7, 1901.

[2] Petersb. med. Woch., 1901, No. 39.

[3] Med. Rec., July 20, 1901.

[4] Münch. med. Woch., No. 40, 1901.

Apelbaum,[1] in discussing the conditions of the **blood in phthisis,** reports a series of uncomplicated cases of that disease, in which he finds that there are **two groups of patients,** even in the early stage of the affection. In most cases the course is very slow, and there is anemia, with reduction of the hemoglobin, the erythrocytes, the leukocytes, and the specific gravity. In the other group, usually well-nourished persons with a fresh color, the blood shows normal conditions. In the second stage of the disease the blood apparently improves; but this is only apparent, for the total amount has decreased and there has been a loss of water. There is, therefore, an anemia which is masked by an oligemia. In the third stage all the elements of the blood again evidently decrease, except the leukocytes, which increase—relatively, at any rate. This decrease is evidently due to a toxic action. The polymorphonuclear neutrophile leukocytes are in excess at this period.

M. D. Bardswell, F. W. Goodbody, and J. E. Chapman[2] report a series of investigations of the **metabolism in cases of pulmonary tuberculosis,** comparing the effects of normal and forced feeding in both pulmonary tuberculosis cases and normal persons. They conclude that pushing the amount of proteids results in an excretion of nitrogen that is out of all proportion to the increased amounts retained. The percentage of urea decreased. Apparently less oxidized forms of nitrogen were excreted in increased quantity. A lessened amount of nitrogen was absorbed, and there was evidence of increased intestinal putrefaction. The absorption of fat remained good, even when very large amounts were taken. The quantity of urinary nitrogen with comparable diets was very small in cases much below weight, and greatest in patients approaching the normal weight. Large diets caused anorexia, dyspepsia, and in some instances vomiting. The dyspeptic symptoms nearly always coincided with figures indicating deranged metabolism. Weight was gained with forced feeding, the gain being greatest on the very large diets; but in such diets the gain seemed to be reached at the expense of the subjective well-being of the patient and of disordered metabolism. Normal generous hospital diets could be distinctly increased with advantage, but a very marked increase had less satisfactory results. In normal persons there was no evidence of any disturbance of metabolism from the excessive dieting; but there was a rapid gain in weight, which was always associated with marked impairment of health, chiefly evidencing itself in gastric and intestinal disturbances. The weight gained was rapidly lost when ordinary diet was resumed. The authors insist that the indiscriminate stuffing of tuberculous patients should be replaced by systematic dieting, the diet to be determined by considering the extent and activity of the disease, the amount that the patient is below weight, his digestive capacity, and to some extent personal dietetic likes and dislikes. There should be especial care in attempting to stuff patients who have reached or passed their highest known weights. Such persons should have a diet about the same as that of a person in good health.

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[1] Berl. klin. Woch., 1901, No. 2.

[2] Brit. Med. Jour., Feb. 22, 1902.

Gidionsen[1] reports the case of a man of 41 with a high grade of emphysema, who had no clinical signs of tuberculosis, but who suddenly **died from profuse pulmonary hemorrhage.** Postmortem showed **numerous varicosities in the trachea;** and near them, just above the bifurcation of the trachea, was a tuberculous ulcer—the only tuberculous change found in the respiratory tract—which had eroded a large vessel and produced the hemorrhage. Owing to the marked emphysema, it had been impossible for the patient to clear his lungs of blood, and he had died with the signs of suffocation. The tuberculous ulceration and the varicosities seemed to be unrelated to each other.

Pratt[2] describes a case of **hemorrhagic purpura associated with general tuberculosis.** The purpura appeared 7 weeks before death, shortly after an attack of epistaxis, hematuria, and melena; and in this case was almost undoubtedly the result of general bacterial infection.

P. Londe and M. Brecy[3] report a case of acute miliary tuberculosis with a general hemorrhagic tendency.

D. D. Stewart[4] reports a remarkable case of **acute splenic miliary tuberculosis** in a woman of 29 years, a nurse who had been in attendance on a patient with tuberculosis. Her illness lasted 68 days. The condition resembled typhoid fever or malaria, but acute miliary tuberculosis was finally considered to be present because all definite signs of the former two diseases were absent. The only notable conditions in the case were high temperature, debility, and marked enlargement of the spleen. At the autopsy the spleen was found to be about four times its normal size and studded with tubercles. There were also miliary tubercles scattered about through other organs. Only one other similar case could be found.

D. Riesman[5] describes a case of **primary tuberculosis of the pericardium** in a man who apparently had previously been healthy. He was taken ill acutely, with pains in the chest, cough, and headache. His pulse was weak; there was a pericardial friction and an exudate in the left pleura. The friction disappeared; the cardiac dulness did not increase. The man's general condition grew worse, and he became cyanotic. Ascites and edema appeared. The pleura was twice tapped; the peritoneal cavity, once. The patient died. The diagnosis had been adhesive pericarditis of tubercular origin, the diagnosis of tuberculosis having been based upon the course of the disease and upon the bloody character of the pleural exudate. The autopsy confirmed the diagnosis, showing adhesive pericarditis in which typical tubercles were found. Guinea-pigs infected with the fluid from the pleural cavity died of tuberculosis. There was an old healed tubercle in the apex of the right lung; but Riesman considers the disease as,. properly speaking, a primary tuberculosis of the pericardium in an anatomic sense, the disease having run the course of pericarditis, and the pericardium having been infected with tubercle bacilli from an unknown source.

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[1] Münch. med. Woch., No. 42, 1901.

[2] Brit. Med. Jour., Sept. 28, 1901.

[3] Gaz. Hebd. de Méd. et de Chir., April 13, 1902.

[4] Am. Jour. Med. Sci., Sept. 10, 1901.

[5] Am. Jour. Med. Sci., 1901.

Plien[1] demonstrated to the Berlin Medical Society some specimens from a woman of 39 who had had the clinical signs of tuberculosis of the intestine. The autopsy showed numerous **tuberculous ulcers, on one of which a carcinoma had developed.**

J. T. Bottomley[2] discusses **28 cases of tuberculous peritonitis.** Three occurred in the first ten years of life, 9 in the second decennium, and 13 in the third. A family tendency to the disease was not a prominent feature. A history of acute abdominal condition preceding the distinctive signs of tuberculous peritonitis was given by 11 patients; in some of the cases tuberculosis of the lungs was present. In 13 instances the peritonitis began acutely; in 13 the onset was slow. Pain was the first symptom in 17 cases; in 8 the earliest symptom was a general feeling of ill health; in 2 it was abdominal distention. In all the cases there were febrile movements of the temperature; a number of them, however, exhibited subnormal temperatures. Pigmentation of the skin, swelling of the inguinal glands, and erythema about the umbilicus were absent in all the cases. All the patients were operated upon. **Eleven regained good health, 11 died, 2 were improved, and 4 passed from observation.** In 19 cases the condition was of the ascitic form; 8 of these recovered. In 7 it was fibrinous; 3 of these recovered. In 2 instances it was associated with ulcerations.

A. Poncet[3] discusses **tuberculous rheumatism or pseudorheumatism.** In the first of the three cases which he reports, a man of 40 had chronic purulent epididymitis. He had previously had prolonged bronchitis and pleurisy. He had for some time had pains and swelling in the joints, which resembled acute rheumatism. The inflammatory signs disappeared in from 8 to 10 days. The pains then localized themselves in the lumbar region and in the right hip. The diagnosis was tuberculosis of the pelvis and Pott's disease. A psoas abscess subsequently developed. The second case, a patient who had tuberculosis of the lung, was suddenly taken with multiple acute arthritis, which soon disappeared and was followed by a fungus suppurating osteoarthritis of the interphalangeal joints of one middle finger. Guinea-pigs injected with the exudate from the joints acquired tuberculosis. This patient also had grating in other joints and a subacute arthritis in the right tibiotarsal joint, and the interphalangeal joints of the right middle finger were completely ankylosed. The third patient had advanced tuberculosis of the lung and a fungous suppurating osteoarthritis of the right ankle. She also presented inflammatory signs in a series of other joints. When she was admitted to the hospital, she had cough and hemoptysis, and multiple joint pains without swelling. Later, swelling of the joints appeared. Autopsy and inoculation of guinea-pigs **demonstrated the tuberculous nature of the joint disease.**

A. Poncet,[4] in referring again to tuberculous pseudorheumatism of the joints, reports a further case as a typical example. A man of 44 who had an almost negative family history, but who was very susceptible to colds himself, had had 2 attacks of what was considered acute articular rheumatism since 1900. In both cases the salicylates and antipyrin were useless. The use of potassium iodid caused the patient to grow worse. Puncture of the joints showed the presence of a fluid, which, when injected into a guinea-pig, produced tuberculosis. The serum-reaction for tuberculosis was also positive. The man afterward developed evident signs of tuberculosis of the lung. Poncet thinks that cases that have the appearance of rheumatism, when antipyrin and salicylic acid are without effect, should be suspected of tuberculosis; and he believes that the serum-reaction will be useful in such cases. Tuberculous pseudorheumatism of various grades—sometimes very mild and evanescent—may, in his opinion, be **the first sign of a tuberculous infection.**

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[1] Centralbl. f. innere Med., No. 14, 1902, p. 360.

[2] Amer. Med., Feb. 15, 1902.

[3] Gaz. des hôp., 1901, No. 85.

[4] Gaz. Hebd. de Méd. et de Chir., No. 86, 1901.

H. Hirschfeld[1] reports 3 cases of **enlargement of the hands and feet of neurotic origin.** Two of them occurred **in association with phthisis,** and the other in association with carcinoma of the esophagus; also because the conditions were somewhat similar to pulmonary osteoarthropathy. An essential difference from the latter condition, however, was in the fact that the enlargement was purely in the soft parts, while in pulmonary osteoarthropathy it is largely of the bones. In one case, to be sure, there was distinct enlargement of the bones, but not those of the hands and feet; only of the joints of the knee and elbow. One case showed a marked tendency to attacks of pain. This patient had symptoms strongly suggestive of tabes dorsalis, and the pains themselves were suggestive of that condition; but the postmortem ultimately showed that there were only scattered areas of degeneration in the spinal cord. The peripheral nerves in this case were not examined. The third case also showed decided tendency to pain, and in this case postmortem revealed distinct degeneration of the nerves. There had also been in this case attacks of edema of the hands and feet, usually occurring with the pains. In the first case, likewise, there had been edema, which, however, appeared only toward the end of the disease, and possibly—though not probably—was due to cardiac weakness. The condition is termed by Hirschfeld "dermatohypertrophia vasomotoria." Since two of these patients showed alterations in the peripheral nerves, the changes in the skin were thought to be due to the nerve alterations. There are changes of the peripheral nerves also in osteoarthropathy.

M. Joseph and G. Trautmann[2] give a discussion of **tuberculosis verrucosa cutis,** based upon their statistics in 26,294 cases of skin disease, venereal disease being absolutely excluded from these statistics. They found 47 cases of tuberculosis verrucosa cutis, 41 of which occurred in men. Nine of them were cabinet-makers, 8 butchers, and 5 locksmiths; the others had various occupations. Of the 8 butchers, 3 cases were of much importance because the patients were engaged at the city abattoir purely in the handling of tuberculous cattle. On investigating the persons working at the abattoir, it was found that of 39 veterinarians and 28 stampers, who came into close relation with tuberculous cattle, none showed skin infection; while the three patients previously mentioned came from 16 laborers connected with the place. The greater frequency of infection in this latter class (laborers) was due to greater carelessness. After a study of the literature the authors come to a positive conclusion that **tuberculosis of animals may certainly be communicated to the skin of human beings.** They consider that there is absolutely no doubt that tuberculosis verrucosa cutis is true tuberculosis. While they admit that the susceptibility of human beings to animal tuberculosis is slight, they insist that such a susceptibility does exist and must be reckoned with; and that, even though skin tuberculosis is relatively harmless, we cannot conclude from this that animal tuberculosis is always nearly harmless for human beings; for human tuberculosis itself exhibits the greatest differences in virulence when it involves different tissues; and the skin is strongly resistant even to human tuberculosis.

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[1] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 3 u. 4.

[2] Deut. med. Woch., Mar. 20, 1902.

M. P. Ravenel[1] reports a further case of tuberculosis of the skin following **accidental inoculation with the bovine tubercle bacillus.** The patient was a veterinary physician who injured himself while performing a postmortem on a tuberculous cow. Four weeks afterward a red nodule developed, which was extirpated. It showed tubercle bacilli, giant-cells, and macroscopically tubercles; and injections into guinea-pigs produced tuberculosis.

P. Baumgarten,[2] in discussing the relation between bovine and human tuberculosis, considers that there is little danger of the transference of the former to man. He refers to some experiments that were carried on 20 years before, by an unmentioned clinician, which have never been published. Patients with malignant growths were injected with cultures of tubercle bacilli in the hope that this might favorably influence the course of the tumors. Since no human cultures were at hand, bovine cultures were used. The patients were neither damaged nor helped, and no tuberculosis resulted. [The investigations of Ravenel and others justify great skepticism regarding the view of Baumgarten. Certainly no one would at the present time feel warranted in carrying out such an experiment as that he relates.]

Diagnosis.—In a discussion of the **diagnostic and therapeutic value of tuberculin** Heron[3] stated that he believed that it was too little used for therapeutic purposes. The chief trouble was that the dose used had been customarily too large and too rapidly increased. He insisted upon its great value in diagnosis. Koch said that he had used it for diagnostic purposes in more than 3000 cases, and had obtained a positive result in more than 99% of the cases. The reaction is more trustworthy in men than in animals. He treats with tuberculin only early cases that are not complicated and are apyretic. In such cases he had often obtained a cure. The treatment ought to be interrupted after some months. When a reaction occurs, some days should be allowed to pass before continuing the treatment. Douglas Powell noted that the number of cases suitable for this treatment is small, because tuberculosis is so often febrile. Denys de Louvain, in animal experiments with tuberculin, had found that tuberculous animals show an increase of resistance under treatment with tuberculin. Otis insisted that the occasional occurrence of a positive reaction in syphilitics greatly restricts the value of tuberculin as a diagnostic agent.

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[1] Univ. of Penna. Med. Bull., Feb., 1902. [2] Berl. klin. Woch., Sept. 2, 1901.
[3] Rév. de la tuberc., Aug., 1901, p. 383.

L. Mazzotti,[1] in discussing the use of tuberculin for diagnostic purposes, states that his experience, after injections in several hundred cases, leads him to consider it, as a rule, an absolute diagnostic method; but **convalescents from typhoid fever frequently give a reaction** when tuberculosis is apparently not present, and it is **questionable whether all chlorotics that react actually have tuberculosis.** The author also refers to the fact that tuberculosis sometimes does not respond with reaction; and he reports the case of a young woman who had suspicious symptoms, but did not react, and, hence, was thought to be free from tuberculosis; but in whom evident tuberculosis appeared soon after. In 2 cases of tuberculosis of the genitals he found that after removal of the local disease the reaction was negative. He thinks, therefore, that tuberculin injections may be useful to surgeons in determining whether an operation had removed all tuberculous tissue. The character of the reaction demonstrates nothing concerning the extent or the probable course of the disease.

Bucco[2] confirms the observations of various French authors that the **injection of an artificial serum** composed of 5 % sodium chlorid and 10 % sodium sulfate produces in tuberculous persons a reaction similar to the tuberculin reaction; but he finds that the same rise of temperature **may be observed in other persons** who are the subjects of various exhausting diseases, such as chronic bronchitis, emphysema, and anemias and cachexias of various kinds.

S. Arloing and P. Courmont[3] report that they have carried out their method of **serum diagnosis in 66 cases,** for the purpose of determining whether certain surgical disorders were tuberculosis or not. They found that of 11 cases which were not tuberculosis, none reacted positively. Of lesions which were clinically tuberculous,—55 cases in all,—41 gave positive reactions and 14 negative. The tuberculosis was situated in various regions—joints, bones, glands, skin, etc. The authors believe that the method of diagnosis is an exceedingly important one in surgical conditions.

Widal and Ravaut[4] have investigated the **agglutination reaction upon the tubercle bacillus exhibited by pleural fluids** of various sources. The technic used was that of Arloing and Courmont. The authors insist upon the importance of using the culture when it is about 5 or 6 days old. They have investigated 24 cases of pleural effusion. In 11, in which the diagnosis was tuberculosis, a positive reaction was obtained 9 times, a doubtful reaction once, and a negative one once. In two pleurisies occurring in phthisical subjects, the results were negative. In 11 pleurisies of nontuberculous nature, there were 11 negative results. The authors recommend the use of the reaction. As to the cytodiagnosis of such effusions, the 11 tuberculous pleurisies had regularly an exceedingly large number of lymphocytes, with almost no endothelial cells. This is, to their mind, an indication of the tuberculous nature of such an effusion. The 11 other cases of pleurisy nontuberculous in origin showed numerous endothelial cells and polymorphonuclear leukocytes, with a relatively small proportion of lymphocytes.

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[1] Clin. med. ital., No. 9, 1901.

[2] Nuova rivista clin.-terapeut., No. 11, 1901.

[3] Rév. de la tuberc., Aug., 1901, p. 380.

[4] Rév. de la tuberc., Aug., 1901, p. 380.

E. Rumpf and L. Guinard[1] have made control tests of the methods of Arloing and Koch for the agglutination of tubercle bacilli. They have investigated 107 patients, 29 of them being in the first stage, 41 in the second, and 37 in the third. The diagnosis was confirmed either by absolutely positive physical signs, by the presence of tubercle bacilli, or by a reaction to tuberculin. The agglutination reaction was positive in 84 % of the cases; in 17, however, only in a dilution of 1:5. They have not tested it in nontuberculous persons. The two methods give results which closely correspond. The injection of the new tuberculin causes a marked increase in the agglutinative powers. The authors' results do not agree with those of other workers in one point: viz., 65 % of the cases in an advanced stage gave a reaction at 1:10 or more. It was generally observed that the **reaction disappeared when the patient was approaching recovery,** 20 of such cases showing, in 8 instances, no agglutination, and in 8 others, only slight agglutination. In severe cases they found, at times, that agglutination was absent; in other instances, when the patients were improving, it was present. The **injection of new tuberculin regularly caused an increase in the reaction when the patients improved under the treatment.** The authors are unable to state whether this change in agglutination indicates that the patient will progress toward a satisfactory recovery or not.

Gebhardt and Torday[2] have investigated the value of the **serum diagnosis of tuberculosis in 176 persons,** using a culture obtained from Arloing. The reaction was obtained in 75 % of the tuberculous, but also in 35 % of the nontuberculous persons. It seemed also positive in 5 normal persons. The authors decide that it is **not suited for clinical use.**

De Grazia[3] has made a series of studies of the serum diagnosis of tuberculosis, and decides that the **reaction is not specific** for that disease, and that this test is of no value in diagnosis or prognosis. The reaction occurs in persons who have no tuberculosis, and in perfectly normal persons; also, the blood-serum of tuberculous subjects causes agglutination of various other microorganisms. The author likewise finds that the reaction **may be extremely slight in tuberculous persons in the early stages of the disease,** at the time when diagnosis is of most importance; but that it shows no relation to the severity of the disease or to its stage, in the intensity of the reaction.

[1] Deut. med. Woch., Feb. 20, 1902.
[2] Ungar. med. Presse, No. 3, 1902.
[3] Gaz. degli osped., 1901, No. 108.

Masius and Belco[1] have made a study concerning the serum-reaction in tuberculosis. They **find the Arloing cultures to be of low virulence** and to be agglutinated by the serum of animals infected with them. If inoculation has not produced infection, the animals do not react. Animals inoculated with more virulent bacilli, which produce disease of rapid course, show but slight agglutinating action. The authors tested a series of patients, with and without tuberculosis, and found agglutination present in almost all of them. In some that had no evident tuberculosis this disease afterward became evident; but in others there was not a suspicion of tuberculosis. The authors believe that the serum-reaction **may help in the diagnosis of tuberculosis, but cannot establish a differential diagnosis.**

Romberg[2] finds that the serum of persons who are free from tuberculosis, as well as that of newborn children, does not produce agglutination of tubercle bacilli. It is doubtful whether the presence of agglutination in the absence of evident tuberculosis should be considered an evidence of the existence of a latent tuberculosis, or not; and whether the absence of agglutination in cases in which tuberculosis seems to be present should indicate the presence of an inactive latent tuberculosis, is also doubtful. In most cases in which tuberculosis is clinically evident the reaction occurs. It is often more marked in the less severe cases; and is frequently absent in very severe, rapid cases, and in cases that are practically cured. The author **considers the serum-reaction to be of no value in the early diagnosis of tuberculosis.** He finds that the degree of alkalescence of the emulsion has a marked influence upon the intensity of the agglutination, and that a still more marked influence is exerted by the extent of dilution of the emulsion.

G. Buard,[3] in studying the serum-reaction in tuberculosis, insists upon the **importance of using constant amounts of culture and serum.** He has also definitely determined the time in which the agglutination occurs, and contrasted it with the maximal time in which identical mixtures with normal serum are agglutinated. **When agglutination occurs in the minimal time, he considers it positive.** Every investigator, therefore, should determine the maximal time for his own cultures. The author finds that blood dried on paper, and even sterilized, retains its agglutinating effect; although the latter is much less active than that of fresh serum. In 50 cases he obtained 48 satisfactory results. In one case of tuberculous pleurisy the result was negative. In a case of Addison's disease, of doubtful nature, the result was positive. **In 25 cases of tuberculosis in children the reaction was always positive.** The serum diagnosis gave negative results in advanced tuberculous cachexia; but in 154 other cases it gave results corresponding with the clinical or anatomic diagnosis [with the exception of the two cases mentioned].

Parodi[4] has investigated the **value of Hesse's culture-medium** for the growth of the tubercle bacillus, and confirms the latter's statement that the bacillus grows very rapidly on this medium, while the pyogenic organisms soon die out. He considers this a valuable method for clinical diagnosis.

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[1] Bull. de l'acad. royale de méd. de Belgique, No. 2, 1902.

[2] Münch. med. Woch., No. 3, 1902.

[3] Gaz. hebd. des sci. med., 1901, No. 30.

[4] Gaz. degli osped., No. 24, 1902.

V. Patella[1] discusses the **cytodiagnosis of exudates and transudates,** with particular reference to the nature of the so-called lymphocytes in the tuberculous exudate. The present article is a preliminary communication only, and is a brief statement of the author's belief that these so-called lymphocytes are really products of endothelial cells: viz., the free nuclei of the latter. He states that the progress of the changes in these cells, up to the point at which the free nuclei are found, can readily be followed; and that, if the case be examined very early in its course, large numbers will be found, while subsequently the pseudolymphocytes will appear in increasing numbers. Cytodiagnosis is, by this observation, made of **much less importance** in his belief, for the **pseudolymphocytes may be found in any condition in which the endothelial cells are cast off in large numbers.**

R. Supino[2] considers **Weigert's stain more valuable for elastic tissue in the sputum than orcein.** For ordinary practical purposes, however, the examination of the first specimen, after adding potassium hydrate solution, is generally sufficient. In 9 cases in which bacilli could not be found, he discovered elastic fibers once; and in 26 in which bacilli were present, elastic fibers were found 19 times.

H. E. Lewis[3] reports a case in which he found **bacilli that were acid and alcohol proof;** that stained well by the Ziehl-Gabbet method; and that, when injected into rabbits, produced suppuration and lymphatic changes, the glands being found caseous. Bacilli were found present in the glands, in the form of moving clumps. The patient had been loading hay and had caught cold, and he and two other men working with him at the same time were taken sick in a similar way. The patient showed no chest signs except moist, bubbling râles over the larger bronchi, and recovered entirely in 4 weeks. Lewis considers that these were **not tubercle bacilli.**

A. Moeller[4] reports that he **was able to grow smegma bacilli on the serum from a blister,** the serum and some of the skin over the blister being transferred to the incubator. He afterward found that these bacilli **grew rapidly on human serum.** He describes their cultural and morphologic characteristics, and refers to a case in which he found numerous acid-fast bacilli in the exudate of acute pleurisy. These bacilli, however, were readily cultivated, grew rapidly, and developed easily at room-temperature; they were, therefore, pseudotubercle bacilli. The patient remained well after a year. In his own sputum the author found, during an attack of bronchitis, numerous small, grayish-white granules, which contained **large numbers of acid-fast bacilli.** He states that he was able to show that these were not tubercle bacilli, and his **health has remained entirely good** for 3 years after this observation.

[1] Deut. med. Woch., April 17, 1902.
[2] Clin. med. ital., No. 9, 1901.
[3] Jour. of Tuberc., Jan., 1902.
[4] Deut. med. Woch., June 26, 1902.

Bozzolo[1] gives an interesting discussion of the **early diagnosis of pulmonary tuberculosis.** The phthisical habitus he considers to be really an evidence of existing infection with tuberculosis, the bacilli probably having remained for a long time in the body without having caused active infection. A **slight temporary albuminuria** in young persons is often a sign of importance in suggesting the occurrence of beginning tuberculosis. It may appear some years before the outbreak of the first distinctive signs. Chloranemia is an exceedingly important sign. The anemia differs from chlorosis in that the hemoglobin is not reduced so strikingly, and in that there is often a slight leukocytosis. There is also usually emaciation. Gastric disturbances are likewise important suggestive signs. The condition of the gastric secretions will usually be found normal or slightly below normal. Bozzolo finds that injections of thyroid extract, in such cases, will increase the HCl of the gastric juice and improve digestion and general nutrition (a rather astonishing result, considering the destructive influence of thyroid upon tissues). The increase in the rapidity of the pulse, without rise of temperature, is also important; and the author considers it of bad prognostic significance. He thinks that a tendency to **sudden outbreaks of sweating** in persons that have not previously been so inclined is frequently an indication of beginning tuberculosis, when it is accompanied by general weakness and pallor. **Pseudorheumatic attacks**—*i. e.*, pains in various joints and tendons—are also suspicious. Dilation of one pupil the author finds to be a rare sign. He insists upon the importance of looking for **localized atrophies of the muscles**—more particularly, those lying near the affected lung, especially the outer portion of the sternocleidomastoid. The atrophy may be shown by reduction in size or by change in mechanical and electrical irritability. The hemoptysis of early phthisis in women is likely to occur during the menstrual period; it is, therefore, frequently confused with hysteric hemoptysis or with vicarious menstruation. The author considers it important, in such cases, to determine whether the heart is involved; if so, this makes the danger of the infection with tuberculosis still greater. The **discovery of an old pleurisy at the apex or elsewhere** is of marked importance in reaching a diagnosis or a prognosis.

C. R. Upson[2] insists upon the importance of **mensuration of the chest** as an aid in the diagnosis of pulmonary tuberculosis, and describes two instruments to be used in assisting mensuration. He considers that in a series of cases reported mensuration had indicated the existence of lung disease before it could be found by ordinary methods.

E. Rondet[3] insists upon the **importance of examining the region of the hilus of the lung** in suspected early tuberculosis. Attention is too exclusively given to the apices. More careful examination of the hilus region would often demonstrate signs when the apices seem entirely free.

Lichtenstein[4] reports the case of a man of 50 who had the signs of an infiltration of the lower part of the left lung; who, 5 years before, had had a prolonged attack of cough, with expectoration; who had since, every 3 or 4 months, had cough with expectoration; and who had once had hemoptysis. The sputum showed no destructive elements; but there were bacilli which had the **specific staining reactions of the tubercle bacilli, except that they were not alcohol-fast;** showed slight morphologic differences; and, when **injected into guinea-pigs, caused no tuberculosis.** The **tuberculin reaction was also negative.** Leyden, in discussion, insisted upon the great importance of such cases in practice, and noted that he had repeatedly observed hemoptysis in cases of recurrent fibrinous bronchitis. He considered that this man's recurrent attacks were fibrinous bronchitis, and that the changes in the left lung were due to a fibrinous pneumonia resulting from the fibrinous bronchitis.

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[1] Rivista critica di clin. med., 1901, Nos. 25–27.

[2] Amer. Med., May 31, 1902.

[3] Gaz. hebd. des sci. méd., 1901, No. 33.

[4] Centralbl. f. innere Med., No. 12, 1902, p. 303.

Memmi[1] discusses the **prognostic value of eosinophilia in tuberculosis.** He examined 35 cases, and found that in a series of instances of beginning tuberculosis eosinophiles were absent; hence, he cannot support the statement that eosinophilia of the sputum precedes the appearance of tubercle bacilli. He found that the eosinophiles were more common in those patients who distinctly improved; but they were not uncommonly present in cases of advanced tuberculosis. Eosinophilia was often marked after hemoptysis. This indicates that their appearance is **not always a favorable sign.**

F. de Grazia,[2] after a study of the **diazo reaction** in tuberculosis, reaches the conclusion that it **has no prognostic value,** and that it is not of importance in determining the existence of a mixed infection.

E. O. Otis,[3] in studying the effect of **tuberculin as a diagnostic agent,** injected 35 cases of definite syphilis, using from 2 to 10 mg. Six undoubted reactions occurred, and there were 5 abortive reactions. The author believes that syphilis in any stage may give a reaction, and that the activity or quiescence of the infection gives no suggestion as to whether the reaction will take place or not. Twenty-six cases of suspected or proved tuberculosis were injected, with perplexing results. Of 8 cases in which the evidence of tuberculosis was considered indubitable, there were 4 reactions and 4 failures to react. In the remaining 18 cases of suspected tuberculosis there were 6 reactions and 12 failures to react. In the latter 12 there was no definite evidence of tuberculosis upon physical examination. The patients were all taken from an ambulatory clinic. [There is no statement as to the possibility that the syphilitic patients that reacted had latent or active tuberculosis.]

Prophylaxis.—Vielefeldt,[4] the head of the German Imperial Life Insurance Department, contributes a discussion of the results that have been obtained from combating consumption with the aid of the **compulsory workmen's insurance in Germany.** As is known, the compulsory insurance agents have erected sanatoriums; have sent the sick to hospitals or health resorts, or put them under the care of private persons; and have in other ways tried, by improving the health of the patients, to benefit not only the latter, but also the financial condition of the insurance companies. The total amount paid out to the families, to keep them properly supplied, in order that the patients might not return to work too soon, was, in 1900, about $125,000. This demonstrates the extent to which the scheme has been carried. Forty-three sanatoriums are in working order, 19 more will soon be opened, and 16 others are planned; consequently there are places for about 20,000 consumptive persons a year. Statistics from the treatment of 21,176 persons are already at hand. **Immediately successful results are said to have been obtained in 68 % to 74 % of the patients.** The beneficial effects lasted but 2 years in 27 % of the men and in 36 % of the women, after the treatment of 1897; while of those treated in 1898, 58 % of the men and 44 % of the women were able to support themselves after 2 years. The general method of determining whether success was obtained was the ability of the patient to support himself by his own labor subsequently.

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[1] Gaz. degli osped., 1901, No. 114.

[2] Clin. med. ital., No. 9, 1901.

[3] Med. News, Aug. 24, 1901.

[4] Brit. Med. Jour., Aug. 24, 1901.

H. N. Barnett[1] states that he has had a considerable experience in the **influence of sea-voyages upon pulmonary phthisis.** He strongly emphasizes, in the first place, the **danger to other passengers** that is always associated with the presence of a tuberculous fellow-passenger. He thinks that it should be demanded that the nature of the patient's disease be known. Probably he should always be forced to take a single cabin; and he should never attempt to travel for his health unless he is in a position to assure himself of comfort during the voyage. As a rule, unless these considerations are fulfilled, the patient suffers much more injury from the journey than he receives benefit from the sea-air.

Bing[2] states that in **examining 12 phthisical patients by exposing slides before them,** he obtained in 3 instances, by subsequent microscopic examination, evidence of the presence of tubercle bacilli. The slides were held at a distance of from 0.5 m. to 2 m.

J. O. Cobb[3] discusses the danger from **unregulated expectoration** by tuberculous persons, and describes a new pattern of pocket sputum-flask, which can be successfully concealed by the handkerchief and can be readily and easily cleansed.

L. N. Boston[4] reports some investigations on tuberculous patients that he has carried out by means of a **special form of mask, carrying a glass slide** 3 inches in front of the mouth and nose. The particular object of the investigation was to see the effect of ordinary breathing and talking, the influence of violent coughing being excluded as far as possible. Specimens collected from 50 patients showed bacteria in 49 instances; 38 of them showed tubercle bacilli in varying numbers. The numbers were the largest in those who had tubercular laryngitis. The detection of bacilli was greatly facilitated by the use of a low-power lens for locating the droplets. The bacilli seemed to be as easily found by this means as by the ordinary method of examination of the sputum. The author insists upon the **importance of these observations in connection with surgical operations.** It is quite possible that any person assisting at an operation while suffering from any acute or chronic infection of the mouth or respiratory tract might readily cause infection of the wound.

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[1] Brit. Med. Jour., Sept. 21, 1901.
[2] Norsk Meg. for Laegevid., No. 11, 1901.
[3] Phila. Med. Jour., April 26, 1902.
[4] Jour. Am. Med. Assoc., Sept. 14, 1901.

B. Fränkel,[1] in a discussion of the prophylaxis of tuberculosis, stated that whether Koch's teaching concerning the difference between human and animal tuberculosis is correct or not, **milk must, as yet, be given boiled, and the meat of tuberculous cattle must be excluded from the diet,** particularly when used raw. He believes that it is extremely important to isolate tuberculous subjects as far as possible, and insists upon the importance of coughing, sneezing, etc., in scattering tubercle bacilli about, and thus causing infection. He has found it impossible to induce patients to use the mask which he has recommended; indeed, most phthisical subjects are even unwilling to take the trouble to hold a cloth before the mouth when coughing, and their relatives and caretakers are often still more indolent. That the danger of infection is marked was shown by the fact that healthy rabbits which were allowed to run about in a phthisical ward in the Charité acquired tuberculosis of the lungs, although they were well fed and the patients all expectorated into cups, and not on the floor. Fränkel insists upon the importance of erecting asylums for the tuberculous in properly situated regions, to which patients can go of their own free will and be well cared for. He directs attention to the fact that if in the whole of Germany 4000 tuberculous persons could be cured in a year in this way, the number of tuberculous persons would be at least 40,000 less after 5 years; for each tuberculous person causes the disease in at least one other person. The decrease in the disease that could be brought about in this way would be very great.

Jacob and Pannwitz[2] insist upon the **importance of the raw meats,** which are so commonly eaten in Europe, **as causes of infection with tuberculosis;** and likewise direct attention to the **danger from nutritive preparations—in particular, meat extracts**—which are now so widely used, and which, because of their method of preparation, are likely to contain living, virulent tubercle bacilli; it is also important to remember in this connection that they are generally used with patients who, because of existing or previous disease, are much more susceptible to infection than robust persons.

S. Arloing[3] discusses the statements of Koch concerning human tuberculosis, and reports some of his own work on the same question. He insists upon the importance of intravenous inoculation. His work was always carried out by this method. The first series was carried out with a bacillus which had been in culture since 1896; and these bacilli, as well as those used in the subsequent experiments, were from a human source. The bacilli in the second series were from a pleural effusion; those in the third were from a case of tuberculous pleuropericarditis, which became generalized before death. The author injected the bacilli into sheep horses, and cattle. In the present paper he reports 13 such injections into animals, which *in toto* make **his positive results amount to 23—4**

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[1] Berl. klin. Woch., No. 38, 1901

[2] Heilkunde, 1901, No. 9.

[3] Bull. de l'acad. de méd., Dec. 24, 1901.

in cattle, 6 in sheep, 10 in horses, and 3 in asses. All these animals are those that were considered by Koch and Schutz to be resistant to human tuberculosis. Arloing then discusses the reasons for the differences in results obtained by him and by Koch and Schutz. The chief reasons are that the latter authors have inoculated altogether too small doses. The result is also dependent upon the fact that the tubercle bacillus, whether of human or of other source, varies greatly in its virulence; and that, consequently, bacilli from various sources should be used in such experiments. He insists that one is **not justified in advising the removal of the restrictions concerning the sale of tuberculous meat and milk.**

F. Löffler,[1] in discussing the **hygiene of dairy products,** states that he agrees with Koch in his belief as to the infrequency, at least, of the transmission of tuberculosis through them. He mentions a number of examples from the literature which convince him that if tuberculosis is ever transmitted from cattle to human beings, the cases are extremely rare. He thinks, however, that our knowledge concerning this point is, as yet, too indefinite to justify us in omitting the regulations which have already been established for the purpose of preventing the spread of tuberculosis.

P. and E. Biedert,[2] referring to the fact that the elder Biedert had stated years ago that tuberculosis of cattle does not greatly endanger human subjects, describe the conditions which they have found in studying the occurrence of phthisis in a region of Bavaria in which the people take milk and milk-products as a large part of their nourishment. These authors found that there was absolutely **no relation between the amount of animal and of human tuberculosis** noted; but that in the four regions in which animal tuberculosis is most frequent, the mortality from that disease among human beings was less than in the other four districts in which there was less animal tuberculosis. They believe that if the influence of cities were excluded, this would be still more evident; and that this question would become clearer if statistics concerning the persons employed in dairies and in the manufacture of dairy products—and particularly statistics from regions in which milk is largely used as food—were collected in large numbers.

A. Moeller[3] makes the statement that he has found a **pseudotubercle bacillus in pearl disease.** He has also made a series of injections of the human bacillus and the pseudobacillus into calves; and finds that the result is about the same in either case, and that neither bacillus produces pearl disease. The conditions produced by the injection of the human tubercle bacillus and the pseudotubercle bacillus into butter are also very much the same. The author considers the organisms producing tuberculosis in the different classes of animals to be closely related, and **believes that the bovine is the nearest to the human disease;** nevertheless, he **thinks that they are entirely distinct.**

Treatment.—J. F. Allen[4] states that **Natal, South Africa, is prac-**

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[1] Deut. med. Woch., Dec. 19 and 26, 1901.

[2] Berl. klin. Woch., No. 47, 1901.

[3] Deut. med. Woch., July 3, 1902.

[4] Lancet, July 27, 1901.

tically free from tuberculosis and is completely free from malaria. There are 3 fairly distinct climates in this district, one being warm, low, and semitropical; another having an altitude of from 2000 to 3000 feet; and the third having altitudes running up to 6000 feet. The native cattle are also free from tuberculosis; although when housed and treated as domestic cattle, they do not exhibit immunity.

Stern[1] insists that the so-called **four-weeks' cure for tuberculosis of the lungs is of great value**; for, by being cared for throughout this length of time in properly regulated institutions, the patients become habituated to the proper methods of caring for themselves. The results of treatment for this period alone are, of themselves, useful; but in a very large proportion of cases the improvement appears afterward, as a result of the care which the patient takes of himself. The cases must, of course, be chosen; rapidly advancing cases and those that show much inflammatory reaction are not suitable.

A. Schenk[2] warmly recommends the treatment of tuberculosis of the lungs by **stagnation hyperemia,** following the plan that has been used by a number of men in the treatment of tuberculosis of the joints. He carries this out by elevating the foot of the bed. The higher the patient can have his feet maintained above the level of his head, the better the treatment is said to work. He puts an ice-cap upon the patient's head and covers his chest with a Leiter's coil or some similar apparatus for increasing the warmth of the chest. Not infrequently, this causes some cardiac weakness; and a systolic murmur may appear, with accentuation of the second pulmonary tone. This, the author thinks, indicates a temporary mitral insufficiency; it can be controlled by the application of cold over the heart. The treatment is said to produce much more ease in expectoration, the sputum collecting in the larger bronchial tubes during the night, and being coughed out easily in the morning.

N. v. Westenryk[3] has investigated the influence of **small amounts of CO_2** upon the bodily temperature. The inhalation of large amounts has repeatedly been studied before, but the influence of the inhalation of small quantities has not been known. The author found that in normal animals the temperature fell; and that if the animals were removed from the apparatus and then replaced in it, the temperature did not again fall, but gradually rose to its former height. After puncture of the heat-center the same effects were observed. He believes that further study of this question is worth undertaking, as it is possible that there may be **some therapeutic value** in the inhalation of small amounts of CO_2 in tuberculosis and other diseases of the respiratory passages.

Cosentino[4] discusses the **mechanism of the cure of tuberculous peritonitis by laparotomy.** He believes, from his experiments and from clinical observations, that it is **due to hyperleukocytosis,** resulting from the operative procedure. He finds that the bactericidal action of the blood-serum is increased after the operation.

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[1] Therap. Monats., No. 10, 1901. [2] Wien. med. Woch., July 6 and 13, 1901.

[3] Arch. f. exper. Path. u. Pharm., Bd. XLVII, Hft. 1 u. 2.

[4] Gaz. degli osped., No. 123, 1901.

Hönig[1] reports a series of cases of tuberculosis that were treated with benefit with **raw meat or raw meat-juice.** He considers the idea that raw meat has some specific antituberculous action to be erroneous, and believes that the treatment produces good results only through improving the general nutrition.

E. Duhourcau[2] discusses the use of the juice of raw meat in the treatment of tuberculosis. He describes excellent results from this in one case, in especial.

B. Rohden[3] recommends **dermo-sapol, a cod-liver oil soap,** as especially suitable for inunctions in scrofulous and tuberculous subjects. He claims that the cod-liver oil is in this way absorbed with great rapidity. The skin should be cleansed, first with alcohol and then with water, and the dermo-sapol should be rubbed in 2 or 3 times a day, 100 grams being used within 10 or 12 days.

H. Claude and A. Zacky[4] report that they have used **lecithin** in tuberculosis with marked improvement in the nutritive condition and in the appetite.

R. Simon[5] discusses the results of **respiratory gymnastics in connection with the inspiration of lignosulfite,** in the treatment of phthisis. He believes that the forcible breathing is of much importance in the treatment of these cases, because it brings the remedy into more immediate contact with the lungs, and because it loosens old pleural adhesions and makes the lungs more movable, increasing their vital capacity and causing the expectoration of any secretion which tends to stagnate in the bronchi. So long as deep breathing causes the expectoration of secretion, catarrh is present, and the treatment should be continued. The author also recommends the continuation of the treatment for some weeks after the secretion has stopped. He states that one may observe the appearance of moist râles for a time after instituting the treatment; this, he believes, is an evidence of the breaking-down and excretion of diseased tissue, as a result of the action of the lignosulfite. He notes that the cavities are cleared out by this method; because of the excretion of their contents, the cavities are likely to appear larger than before. He finally states, however, that in advanced cases, when the patients are already weak, this treatment is likely to be depressing, the respiratory exercise itself being a strain for such persons; in such cases, therefore, it should not be used.

E. Franck[6] discusses the **hetol** treatment of tuberculosis, reporting 18 cases in which this was used by intravenous injection. This method he considers by all means the most satisfactory, except in those cases in which, because of extreme weakness, etc., it is difficult to inject directly into a vein. He states that in about 500 injections he has never seen any unfavorable symptoms; consequently he does not consider this treatment at all dangerous. Of the 18 patients, 5 were treated but a short time; the other 13 were given, in all, 408 injections. Five of them were severely ill; the other 8 were in the incipient stage. The former were but little benefited; of the latter, 6 were practically cured and 2 decidedly improved.

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[1] Klin.-therap. Woch., No. 30, 1901.

[2] Zeitschr. f. Tuberc. u. Heilstätten, 1901, Hft. 3.

[3] Therap. Monatsh., No. 8, 1901.

[4] La Presse méd., Sept. 28, 1901.

[5] Therap. Monatsh., 1901, No. 11.

[6] Therap. Monatsh., Dec., 1901.

Anelli[1] claims that he has had excellent results, in the treatment of phthisis, from the **intravenous injection of sodium cacodylate,** using about $\frac{3}{4}$ of a grain at a dose.

Robin and Binet,[2] in a discussion of the treatment of phthisis, describe their studies of the respiratory interchange, and insist that the most notable characteristic of phthisis is the tendency of the organism to fix an increased amount of oxygen as carbon dioxid. They consider, therefore, that the preliminary symptoms of tuberculosis are due to irritability and increased vitality, rather than the decrease of vitality. From this standpoint, they claim that arsenic is harmful, because it tends to increase tissue-change. They think that by studying the respiratory interchange it may be determined whether drugs are likely to have a good or a bad effect in phthisis.

A. Blumenthal[3] has made a study of **pyramidon and its camphor and salicylic salts** in the treatment of phthisis, and decides that the salts have as satisfactory an effect as the pyramidon itself. The dose which he used was from 25 to 45 grains a day.

W. Lublinski[4] found that pyramidon had a good effect upon the fever in most cases of phthisis, but that, for reasons which were not at all clear, it was in some cases without effect, and in other instances produced little result.

A. Nolda[5] reports 8 cases of phthisical **night-sweats treated with tannoform,** used as a powder, mixed with 2 parts of talcum, and rubbed into the portions of the body which most commonly sweat most freely. In 7 cases the sweating disappeared, and the eighth case was much improved.

A. Lozecki[6] discusses the use of **parachlorphenol** and **menthorol** in the treatment of tuberculosis of the upper air-passages. The former is useful as a caustic and disinfectant, but has a very unpleasant taste and odor; the menthorol, which is a mixture of parachlorphenol and menthol, has not this disadvantage. Used in a 5% up to a 15 % solution, and applied directly, it is said to produce good results and to have no disadvantages.

H. Harper[7] reports a series of cases of tuberculosis treated with **urea,** and states that he believes that urea has a specific action upon this disease that is quite as marked as that of mercury upon syphilis. He considers that it is a stimulant, and that it causes no increased excretion of urea through the kidneys in tuberculosis.

A. H. Buch[8] reports his results from the treatment of tuberculosis with urea, claiming excellent effects. [This treatment seems to be based upon a wholly erroneous idea of metabolic conditions and also upon some very inconclusive experiments with the use of unsatisfactory methods.]

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[1] Rif. med., July 19, 1901.

[2] Bull. de l'Acad. de Méd., No. 3, 1902.

[3] Deut. Aerzte-Zeitung, 1901, Hft. 19.

[4] Therap. Monatsh., No. 10, 1901.

[5] Berl. klin. Woch., July 21, 1901.

[6] Therap. Monatsh., Jan., 1902.

[7] Lancet, Dec. 7, 1901.

[8] Phila. Med Jour., Sept. 14, 1901.

F. Jussen[1] discusses the results that he obtained in pulmonary tuberculosis with **tuberculocidin Klebs.** Fourteen cases in the first stage showed, in every instance, sufficient improvment to allow the patients to return to their work. This also occurred in 6 of 11 cases in the second stage, and in 3 of 11 cases in the third. The author believes that the improvement was evidently largely due to the tuberculocidin, and that this substance ought to be tested further. He considers that it is certainly not diluted tuberculin. It was noteworthy that in cases with mixed infection there was a marked tendency to a reaction with fever, and that in a number of instances hemoptysis followed the use of the tuberculocidin. Jussen insists that it **cannot be considered a wholly harmless substance** if there is mixed infection.

Figari and Lattas[2] report the results which they have obtained from the use of **Maragliano's serum.** In all, there were 171 cases, which did not include very early ones; 44 were considered to have been cured, 76 were improved, 39 remained stationary, 12 grew worse, none died. The cases were those that had been under treatment from January 1, 1900, to June 1, 1901.

Monteverdi[3] reports 3 cases of tuberculosis which he treated with Maragliano's serum. The symptoms, particularly the fever and the night-sweats, were greatly improved.

O. Niedner[4] reports a number of cases of **hemoptysis** which he has treated by first determining the side from which the bleeding came, and then very **firmly strapping this side of the chest,** passing the straps first over the apex and then around the lower part of that side of the thorax. He believes that the results are quite as good as with gelatin; that the procedure is free from danger, while the use of gelatin is not; and that it can be satisfactorily carried out anywhere, without apparatus.

Grunow[5] discusses the use of **gelatin injections** in hemorrhage. He believes that the injections were regularly beneficial in hemoptysis from phthisis, and that they caused cessation of the hemorrhages in gangrene of the lung, hematemesis, and renal hemorrhage. The collateral unfavorable effects are pain, which may be severe; fever; urticaria; and deep-seated swelling. The author combines the use of gelatin with the administration of morphin and other styptics.

A. G. R. Foulerton[6] gives a general discussion of the influence of secondary infections in chronic phthisis. He believes that secondary infection may be largely controlled by treatment, and that the **climatic treatment** of the disease—particularly high altitudes—acts largely by controlling this secondary infection. He also considers that the recrudescences of the disease in those cured by climatic treatment are chiefly due to secondary infection.

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[1] Centralbl. f. innere Med., June 7, 1902.

[2] Gaz. degli osped., No. 117, 1901.

[3] Gaz. degli osped., No. 96, 1901.

[4] Deut. med. Woch., June 5, 1902.

[5] Berl. klin. Woch., Aug. 12, 1901.

[6] Phila. Med. Jour., Nov. 2, 1901.

DIATHETIC DISEASES.

PENTOSURIA.

F. Meyer[1] notes the **importance of distinguishing cases of chronic pentosuria from diabetes mellitus.** Pentosuria is uncommon, but is very likely to be mistaken for diabetes, because of the marked reaction to copper tests. The author describes a case in which an erroneous diagnosis of diabetes was made, and in which it was afterward stated that the patient had been cured, on account of the fact that polarimetric examination of the urine was negative. Copper tests were afterward again positive, but fermentation and polarization were negative and the orcin test was positive. The patient had become decidedly emaciated; there were marked neurasthenic symptoms and severe neuralgias. Strict diet, the Carlsbad cure, moderate control of the carbohydrates, or an **absolutely unlimited diet, had no effect upon the excretion of pentoses,** but with a diet consisting chiefly of 2 liters of milk and 50 grams of somatose a day, the weight increased and the neuralgia disappeared.

M. Bial[2] reports a **new method for determining the presence of pentoses** in the urine. It consists merely in the addition of ferric chlorid to the orcin-hydrochloric acid solution. Of this mixture 4 or 5 cc. is added to 2 cc. or 3 cc. of the suspected urine. The glass is closed with cotton, and is held over the flame until the first bubbles form, when the test at once becomes positive. The author says that ferric chlorid also readily splits off glycuronic acid from its compounds in the urine; and that, by boiling about one minute, glycuronic acid may be determined to be present. This test will also show the presence of glycuronic acid in normal urine. Bial states that this is a rapid and certain method for determining the presence of pentoses and glycuronic acid. [He does not, however, make it clear how to distinguish one from the other, nor does he explain whether the test reacts only to these substances in the form in which he uses it.]

K. v. Alfthan[3] states that he has determined that **pentoses may be definitely demonstrated to be present** in the urine, even in the presence of glycuronic compounds, by the following method: 500 cc. of urine is treated with benzoyl chlorid and sodium hydrate; and the esters are afterward saponified with sodium ethylate, and the fluid filtered. The phloroglucin-HCl or orcin-HCl test may then be applied. If positively definite, it indicates the presence of pentoses; since glycuronic compounds formed are insoluble, and, therefore, do not pass into the filtrate.

GLYCOSURIA.

J. Donath and W. Schlesinger[4] have made a study of the **blood-sugar in alimentary glycosuria in dogs.** They state that when amounts of sugar so large that the organism cannot assimilate them are given to dogs, the kidneys exert a regulatory action, excreting the excess of sugar and preventing a notable degree of hyperglycemia. In some cases, however, the kidneys do not exert this action, even when apparently normal; and in these cases a hyperglycemia occurs. In rarer cases sugar is excreted, and yet hyperglycemia occurs, probably because the assimilation of sugar is so disturbed that the kidneys are unable to compensate for the difficulty. When neither glycosuria nor hyperglycemia occurs, the authors consider that there is either a very slow absorption of sugar or an excessive power of assimilating it. They think, therefore, that a **negative result in an examination for alimentary glycosuria does not offer absolute testimony that the sugar-assimilating powers are normal,** because different individuals have marked differences in the power of the kidney to excrete or to hold back the sugar.

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[1] Berl. klin. Woch., No. 30, 1901.

[2] Deut. med. Woch., April 10, 1902.

[3] Arch. f. exp. Path. u. Pharm., Bd. XLVII, Hefte 5 u. 6.

[4] Wien. klin. Rundschau, No. 41, 1901.

K. Reuter[1] discusses the literature and his own experiences concerning the **influence of the ingestion of alcohol** and carbohydrates in the production of glycosuria. He believes that glycosuria can be caused in this way, and that the assimilative power for carbohydrates can be so reduced that a glycosuria may persist for some time after the use of alcohol has been stopped. He thinks, however, that this condition is not a very unfavorable one, although it is a pathologic state. There are no reports which demonstrate that the condition actually goes on to true diabetes mellitus. The excretion of sugar ceases after the patient is put on a strict diet, and acetone and diacetic acid do not appear in the urine. Rest and a strict diet cause the assimilative powers to approach to the normal again; and if the use of carbohydrates is taken up slowly, the patient usually becomes able to take 100 to 150 grains of glucose without any glycosuria. Strict diet and the gradual addition of carbohydrates should be carried out until the urine remains free from sugar after using the above-mentioned amount of glucose. The author believes that the alcohol **produces its effect by action upon the cerebrum and the basal ganglia.**

K. Walko[2] discusses the conditions in **141 cases of phosphorus-poisoning** observed in v. Jaksch's clinic, with especial reference to the occurrence of **spontaneous or alimentary glycosuria and acetonuria.** Spontaneous glycosuria was found in only 6 cases. Alimentary glycosuria (after 100 grams of glucose) was present in 61 cases. The cause of this reduction in the power of assimilating glucose may be found in anatomic or functional changes in the liver. [In such a general disturbance as phosphorus-poisoning it can hardly be said directly that the liver alone is at fault in this particular, especially in consideration of the large amount of work that has been done to show that liver diseases *per se* do not seem to have a marked tendency to produce alimentary glycosuria.] The author believes that these observations indicate a very direct relation between fatty liver and glycosuria. [The criticism just made applies to this conclusion also.] Alimentary glycosuria, as a rule, appears during the first 12 days of the poisoning, and is fairly proportionate to the severity of the case. **Acetonuria commonly appears** at the beginning of the disease, may be persistent or temporary, and has no direct or striking relation to the severity of the case or to the occurrence of alimentary glycosuria.

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[1] Mittheil. aus den Hamburg. Staatskrankenanstalten, 1901.

[2] Zeitschr. f. Heilk., Hft. 9, 1901.

Marie des Bouveries[1] contributes an interesting paper concerning **glycosuria in pregnant and puerperal women.** In 103 pregnant women she carried out 353 examinations, and found **109 positive glucose reactions.** Wormmüller's reaction was used, with the demonstration of the osazone. In other words, she found glucose in more than 40 % of the cases, and in a slightly larger number of multiparas than of primiparas. There were but few positive results in the puerperal. Some of these had shown no glycosuria during pregnancy, however. Of the 131 puerperal urines, only 9 gave a positive reaction. One hundred grams of glucose, cane-sugar, or lactose were given on an empty stomach. Nonpregnant women rarely reacted; but of 24 pregnant women, 16 showed glycosuria; and lactose passed into the urine even more easily. Small amounts of lactose were also frequently found in the later months of pregnancy, and **lactose was constantly present** in the urine of puerperal women throughout a long period. The reason for the disappearance of the glucose directly after the labor could not be determined.

Salomon[2] reports 2 cases in which there were **fatty stools in patients who had transitory glycosuria,** and were believed to have pancreatic disease. He **used pancreatin** from one source without result. He afterward used pancreas substance and pancreatin from another source, with the result that the fat-content of the stools was greatly decreased and the nitrogen-absorption increased. The same result was obtained with a preparation called pankreon—a combination of pancreatin and tannin, which is recommended because it is less influenced by the gastric juice than are the other preparations.

Suprarenal Glycosuria.—T. Blum[3] reports that **suprarenal extract from various animals,** and in one case from a man, when sterilized, filtered, and **injected into animals, caused glycosuria.** He gave the injections subcutaneously, in order to avoid, as far as possible, the marked increase of blood-pressure produced by intravenous injections. The glycosuria was more marked when the specimens were not subjected to heat. **Feeding with suprarenal did not produce these results.** The result was obtained in 22 cases. Glycosuria occurred when the diet contained no carbohydrates; and one dog had been starved for 19 days, and glycogen had probably disappeared entirely from the liver in this time. The author thinks that it is possible that there is a suprarenal diabetes, and that the glycosuria is produced by the toxic action on one or more organs concerned in carbohydrate metabolism. The glycosuria is **not due to alterations in blood-pressure,** because the effect upon blood-pressure is past before the glycosuria reaches its more marked stage, and the glycosuria lasts for many hours afterward. He notes that bile-pigment appeared in the urine of some of the animals, and that this perhaps indicates that the liver is chiefly affected, although this may be only a coincident action upon the liver. There was no relation between the excretion of bile-pigments and the amount of glycosuria, and bile-pigment appeared only rarely. **Acetone and diacetic acid were not found.** The site of injection often became infected, possibly as the direct result of the local action of the extract. He thinks that entire failure of the function of the suprarenal glands produces Addison's disease; while, if the glands carry out their function imperfectly, but do not entirely lose it, we have diabetes.

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[1] Diss. Amsterdam, 1901.

[2] Berl. klin. Woch., No. 3, 1902.

[3] Deut. Arch. f. klin. Med., Bd. LXXI, Hefte 2 u. 3.

G. Zuelzer[1] **confirms the work of Blum,** reporting experiments on 5 dogs, 5 rabbits, and 4 cats, all of which showed glycosuria after suprarenal injections, the glycosuria lasting from 24 to 48 hours, and occurring in animals whose food had contained no carbohydrates. A recurrence of the glycosuria could be produced by a renewed injection. The skin showed local changes about the site of injection, particularly loss of hair, thickening, and scaliness. The author occasionally found albumin in the urine, repeatedly discovered albumoses, and in a very few instances noted a ferric-chlorid reaction. He extirpated the kidneys, determined the sugar-content of the blood, and then injected suprarenal and again determined the blood-sugar. In 2 of 3 cases there was an **increase of sugar in the blood,** and in the third case the negative result was probably due to loss of blood in the operation. Zuelzer thinks that **the glycosuria is not of renal origin.** He found that **levulose occasionally appeared in the urine** when fed to these animals. Grape-sugar, administered to them, was excreted in as great a proprotion as 25 %. Milk-sugar was also excreted. He injected 2 cats repeatedly, throughout a period of 4 weeks; and notes that a marked pigmentation appeared on the lips, the nose, and the mucous membranes of the mouth. This pigmentation disappeared some weeks after the injections were stopped.

Herter and Richards[2] report a series of experiments concerning adrenalin glycosuria. They found that, when **given intraperitoneally, it very rapidly produced a marked glycosuria,** the percentage of sugar reaching as high a point as 9.17. This glycosuria is not dependent upon the presence of a diastatic ferment, stored in or formed by the suprarenal gland; because adrenalin is prepared at a temperature sufficient to destroy such a ferment, and the solution may be boiled before using and yet cause glycosuria. At times, adrenal injections produce destructive lesions of the gastrointestinal tract and the pancreas; and the authors, after a fatal dose, in one instance, found that the cells in the **islands of Langerhans were the seat of granular degeneration,** the nuclei showing a marked loss of chromatin substance. In some regions the islands of Langerhans seemed to be more injured than the surrounding cells. Intraperitoneal injections caused a much more marked effect than subcutaneous injections. The authors also noted an extremely rapid appearance of sugar in the urine after painting adrenalin solution upon the pancreas with a soft brush. They state, also, that adrenalin may be added to a solution of glycogen, and that no change occurs in the glycogen after 24 hours.

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[1] Berl. klin. Woch., No. 48, 1901.

[2] Med. News, Feb. 1, 1902.

DIABETES MELLITUS.

Etiology.—A. Pallatschek,[1] discussing the etiology of diabetes, refers particularly to the generally accepted statement that **Jews are more subject to this disease than other races.** In 10 years he states that he has had 4719 patients under his care in Carlsbad, 2380 of whom were Christians, 2383 Jews, and 5 Mohammedans. Of these, there were 653 diabetics—289 Christians and 364 Jews; hence 13.8 % of the total were diabetics, and out of each 1000 of the Christians 124 were diabetic, while out of each 1000 of the Jews 155 were diabetic. This slight difference he attributes to causes other than racial; for example, 4 fatal cases occurred; 3 of these were Christians and 1 a Jew. He has found in Jews almost all the severe complications that he has observed in Christians. These facts, he believes, indicate that Christians seek places such as Carlsbad later in the disease than the Jews, and he believes that of the Christians who have diabetes a relatively smaller number come under observation than is the case with Jews. Hence he considers that **Jews are not really more subject to the disease,** the difference in number being merely due to the difference in their habits of consulting physicians.

R. Lépine,[2] in referring to the possible existence of **diabetogenous leukomains,** states that the injection of an alcoholic extract of the blood of a normal dog caused a transitory glycosuria only, while the extract from the blood of a dog whose pancreas had been removed provoked a glycosuria which lasted several days. He believes that this indicates that he was probably right in his statement that the **pancreas destroys diabetogenous toxic substances.**

H. Strauss,[3] in an article on alimentary levulosuria, speaks very positively concerning his views as to the existence of a **hepatogenous form of diabetes.** He finds that patients with liver disease react to the ingestion of large amounts of levulose by a levulosuria, and not by a dextrosuria. He had previously reported a series of cases of liver disease in which alimentary dextrosuria was absent, and he now describes a further series of cases in which the same conditions were present. He states that from a study of the literature he can find **only about 2 % of cases in which alimentary dextrosuria was reported,** and that in these cases there is some doubt about the result, because cane-sugar was occasionally used, and this contains levulose. As to the alcoholic form of diabetes, which is sometimes said to occur, and which is believed to be produced through the influence of alcohol upon the liver, Strauss refers to his own previous report of 58 cases of chronic alcoholism, in which he found alimentary glycosuria in only 5 %. He now reports 100 further cases, and refers to J. Strauss's 30 cases, in all of which the test for alimentary glycosuria was negative with the exception of two instances; and in these two cases there were complicating conditions. The author believes that Strümpell's results in beer-drinkers are probably attributable to the carbohydrate contained in the beer, or to the carbonic acid or spices, and not to the alcohol. He has had a **series of beer-drinkers examined concerning alimentary glycosuria,** having them take a liter of beer containing 60 grams of carbohydrate in solution, on the empty stomach. In 32 instances the test was regularly negative, and in two instances 120 grams of carbohydrate were taken without the excretion of any sugar. Strauss, therefore, believes that there is **no evidence of the existence of a hepatogenous form of diabetes,** and he also thinks that we **must be careful in saying that alcohol produces diabetes,** as there is no distinct evidence that it does.

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[1] Zeitschr. f. klin. Med., Bd. XLII, H. 5 u. 6.
[2] Berl. klin. Woch., April 21, 1902.
[3] Deut. med. Woch., Nov. 7, 1901.

L. Popielski[1] contributes a study concerning the **nerve-center of the pancreas** that produces secretion, which may prove of importance in connection with the various diseases of the pancreas, and in relation to diabetes. The author finds that the reflex secretory center is situated in the nerve-ganglion of the gland itself.

S. Flexner,[2] in discussing the various theories of the causation of diabetes, states that it must be considered that **the symptom-complex is not dependent upon any one primary set of functional or anatomic conditions.** He refers to his work with Pearce, in which they found glycosuria 15 minutes after the injection of gastric juice into the duct of Wirsung in dogs. The organs which seem to have an established place in carrying out carbohydrate metabolism are the pancreas and the liver, but the mechanism of their action has not been explained. Nervous influence is of great importance. It is probably not specific, but is the ordinary trophic influence.

F. Hirschfeld[3] discusses the **relation between accidents of various kinds and diabetes,** and strongly insists that accidents are very active in the production of this disease. He refers to a number of cases in the literature which seem to him to indicate this fact strongly, but he offers no very definite testimony. He believes that accidents probably cause pancreatic changes more frequently than is usually believed, and furthermore states that even when pancreatic disease is found in diabetes, if the history is properly examined into it will often seem probable that the actual exciting cause of the diabetes was really an accident. In such cases pancreatic disease constitutes a predisposing cause of the disease, while the **accident is the immediate cause.**

O. Loewi[4] reports as his results from some experimental work, that the **difference in the effects of phloridzin when injected subcutaneously and when given by the mouth** is due to the fact that phloridzin is not entirely absorbed when taken into the gastrointestinal tract, and that a cleavage occurs, in which there is evolved a product that causes a decided glycosuria, but that, under ordinary circumstances, is absorbed only with difficulty and is largely lost in the feces. He also shows that phloridzin, when injected into the mesenteric vein, and when, therefore, it necessarily passes through the liver, has quite as active an effect as when injected subcutaneously; hence, the **liver does not seem to be**

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[1] Pflüger's Archiv, Bd. LXXXVI, p. 215.

[2] Univ. of Penna. Med. Bull., Jan., 1902.

[3] Deut. med. Woch., Aug. 22, 1901.

[4] Arch. f. exper. Path. u. Pharm., Bd. XLVII, H. 1 u. 2.

active in causing the difference in effects when the two methods of administration are used. The author also found that the dose of phloridzin which will produce the most marked excretion of sugar is not the same with all kinds and amounts of food; and that, if the sugar-producing food is increased, the dose of phloridzin must be increased in order to get the maximum effect. Change in the body-temperature was without notable influence upon phloridzin glycosuria.

E. v. Czyhlarz and W. Schlesinger[1] have carried out a series of **estimations of the blood-sugar in phloridzin diabetes,** and state that of 22 estimations, 12 showed normal values—3 of them, indeed, being at the upper border of the normal. Subnormal values were found in the other cases. When the glycosuria is prolonged, the blood-sugar value sinks. This the author considers to be due to the organism having been gradually robbed of its carbohydrate store. As a result of these studies, they hold to Minkowski's theory that the **phloridzin acts as a vehicle for the excretion of the sugar.** The presence of normal or excessive blood-sugar values is entirely in accordance with this theory, unless one considers that the splitting of the sugar from phloridzin and the excretion occur at the same time and in the same place in the kidneys. One can, however, readily conceive that the kidney can split the sugar from the phloridzin without at once excreting it. The sugar then lodges in the circulation; and normal, or even excessive, amounts of blood-sugar may be found present.

O. Loewi,[2] in discussing the question of the **production of sugar from fat,** reports some experiments in which, in phloridzin diabetes, he administered large quantities of fat and demonstrated that the sugar excretion was not increased. The nitrogen excretion, as well as the **sugar excretion, usually fell.** This was probably attributable to the fact that the **fat acts as a protective against albumin disintegration;** consequently, the sugar ordinarily produced from the albumin disintegration was reduced in amount. The nitrogen-excretion, however, was influenced much more markedly than the sugar-excretion; and the author believes that we must consider the possibility that, **in the destruction of albumin, its various elements may take various pathways,** and, in particular, that a nitrogen-containing portion may be retained, while another portion—especially the carbohydrate element—may be excreted or oxidized in the body. He also believes the fat first acts in protecting albumin when the albumin molecule has already undergone cleavage and the carbohydrate group is split off. He particularly insists that the excretion of nitrogen does not always run parallel with the disintegration of nitrogenous tissues, and that we are not justified in assuming that a rise in the ratio of dextrose to nitrogen means that there has been an increase in the production of sugar; it may also mean that there has been a retention of the nitrogenous element or that the difficulty in assimilating sugar has increased. His experiments showed absolutely **no indication that sugar can be produced from fat.**

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[1] Wien. klin. Rundschau, No. 41, 1901.

[2] Arch. f. exper. Path. u. Pharm., Bd. XLVII, H. 1 u 2

Umber,[1] discussing the relation between the excretion of sugar and that of nitrogen, with destruction of tissue, in diabetes, directs especial attention to the fact that when nitrogenous tissue or food is destroyed in the body in that disease, the greater part of the sugar yielded is eliminated; while an uncertain portion of the nitrogen is retained, and produces tissue. Hence, he considers it **impossible to draw any definite conclusions as to the amount of sugar yielded by protein food through observing the relation between the sugar and the nitrogen excreted.** The amounts of protein substances which produce a given quantity of sugar are undoubtedly larger than has usually been maintained. It is impossible to determine how much sugar is yielded by nitrogenous substances in the body, as long as we know so little as we do about the synthetic production of sugars and of protein substances therein; and the study of these substances is especially difficult, because synthetic products have certainly—in many instances, at least—only a very temporary existence, and are almost immediately broken down or built up into other substances.

A. Hesse[2] gives an interesting discussion of **nitrogen-metabolism and sugar-excretion in severe diabetes,** his most important point being the conclusion, from his own studies and from the work previously done by Umber and others, that the **dextrose-nitrogen ratio is valueless,** and should disappear from the discussion of the question as to whether sugar is ever formed from fat. He insists that we must believe it likely that in diabetes, at any rate,—and probably in other conditions,—the carbohydrate element of protein tissue or food may be split off and either oxidized or excreted as sugar, while the nitrogenous element is retained. If this is the case, it is at once evident that the dextrose-nitrogen ratio means nothing, and that, so far, there is **no real testimony that sugar can be formed from fat.** It also proves that the **urinary nitrogen is not a real indication of the amount of protein destroyed.** The author also directs attention to the fact that with Lipliawski's reaction he was able to demonstrate the presence of diacetic acid when any considerable amount of acetone was present, and when the ferric chlorid reaction was frequently negative. A case which he studied also indicated that a **large amount of protein food does not produce acetonuria, while a diet rich in fats and poor in carbohydrates does** rapidly produce it.

T. Rumpf[3] contributes an extensive paper concerning his investigations in a number of cases of diabetes. He particularly notes the high dextrose-nitrogen ratio that he found in the cases reported, and believes that this indicates positively that sugar must have been formed from fat; although, at the same time, he emphasizes the fact that this probably takes place only in extreme instances and in comparatively small amounts. [He entirely overlooks the probability discussed by Hesse and others, above.] He also insists that it is **impossible that**

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[1] Therap. der Gegenwart, 1901, No. 10.
[2] Zeitschr. f. klin. Med., Bd. XLV, H. 3 u. 4.
[3] Zeitschr. f. klin. Med., Bd. XLV, H. 3 u. 4.

acid intoxication alone should be the regular cause of diabetic coma; since the degree of the former when the latter comes on may be comparatively slight, and, on the other hand, a marked acid intoxication may persist a long time without any sign of coma. [In this point the author overlooks the simple probability that individuals vary greatly in their resistance to acid intoxication.] He believes that one of his cases in particular demonstrates that a **loss of fluid from the system is one of the most important—and perhaps the most important—of the causes of coma.** He especially directs attention, also, to the rapid appearance of signs of intoxication when he attempted, in his studies, to exclude carbohydrates, to increase the fats, or to reduce the amount of proteid taken below the normal point. He insists that it is essential that carbohydrates shall not be absolutely withdrawn from the diet in severe cases, and that the proteids shall not be greatly reduced. He states that he **has stopped using intravenous infusions of sodium bicarbonate** in the treatment of coma, believing that this method is useless.

L. Mohr[1] presents a study of a case of diabetes. He decides that his work and that of others show that in severe cases of diabetes **sugar may be formed from fat.**

O. Loewi[2] has investigated the correctness of the statement of P. Mayer that glycuronic acid is a product of the oxidation of glucose. His method was to produce phloridzin diabetes in animals, and then to administer large doses of camphor, which carries off a good deal of combined glycuronic acid. If glycuronic acid is a product of glucose, a marked increase in the glycuronic acid excretion should reduce the sugar-excretion proportionately. The author's results were to demonstrate that camphor causes a decrease in sugar-excretion, but one which seems to be entirely independent of the amount of glycuronic acid present. It is also **highly probable that the mother-substance of glycuronic acid is not the same as that of sugar,** and that the **glycuronic acid does not come from the oxidation of glucose.** This has already been made extremely probable by Emil Fischer's statement that the aldehyd group of the sugar pairs with other substances, to be excreted as paired glycuronic acids, the pairing taking place before the sugar molecule is oxidized at all.

D. L. Edsall,[3] in a discussion of the **clinical significance of glycuronic acid,** opposes the view expressed by Mayer, that an excess in the excretion of glycuronic compounds indicates a reduction in the oxidative processes. With the latter, he has found an excess of glycuronic compounds in diabetes (4 of 8 cases); in typhoid fever (6 of 6 cases); and also in general sepsis, severe tonsillitis, and other infectious conditions. He believes, however, that the **increase in the glycuronic compounds is chiefly an indication of intoxication,** and is merely a method of protecting the system from poisoning, through causing the excretion of toxic substances in harmless form. He thinks it possible that the determination of the presence of an excess of glycuronic compounds in the urine may be a method of demonstrating the presence of intoxication in such conditions as hysteria, neurasthenia, and other states in which intoxication is suspected, but otherwise not proved to be present. He has found a marked orcin reaction in a number of such cases, when all evidences of gastro-intestinal disturbance were absent. He does not believe that Mayer is correct in stating that the development of diabetes may be anticipated through the discovery of an excess in glycuronic-acid excretion. The orcin test is, however, valuable, because it provides a rapid method of demonstrating the cause of the confusing reaction to copper which is obtained when an excess of glycuronic compounds is present. Mayer considered that the increase in the benzoyl esters in the fermented urine in diabetes mellitus, which had been demonstrated by Rosin and v. Alfthan, was due to increase in glycuronic compounds. Edsall estimated the benzoyl esters in 3 cases of diabetes in which there was no orcin reaction for glycuronic acid. In these 3 cases the **esters were very largely increased.** He consequently thinks that their increase is not necessarily related to an increase in the glycuronic acid, and that it **probably indicates a general disturbance of carbohydrate metabolism.** This is further testimony that **diabetes cannot be explained through alterations in glycolysis.**

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[1] Berl. klin. Woch., Sept. 9, 1901.
[2] Arch. f. exper. Path. u. Pharm., Bd. XLVII, H. 1 u. 2.
[3] Univ. of Penna. Med. Bull., April, 1902.

H. Rosin[1] gives an interesting review of the work on the etiology of mellliturias, and then refers to some work by Spaeth undertaken under his direction, which showed that the **administration of large amounts of carbohydrates to dogs causes the appearance in the urine not only of glucose, but also of other unknown carbohydrates.** A decrease of the carbohydrates, with increase of protein, decreases the amount of these substances; and on a fat diet their amount is least of all. The author insists that there should be more accurate study of other carbohydrates than glucose in diabetes.

E. Bendix and A. Bickel,[2] in **discussing the teaching concerning glycolysis, direct attention to 3 kinds of error in technic :** (1) Errors in the method of determining the amount of sugar; (2) errors due to bacterial action; (3) errors due to destruction, or apparent destruction, of the sugar by purely chemical processes. In some of the work that has been done **bacterial action** has probably been satisfactorily excluded, but in most of the work it has not. The **methods used to determine the sugar are not satisfactory,** and there is a constant error, of greater or less degree, in this point. **Chemical changes have been largely overlooked.** The authors call attention to the fact that Lobry de Bruyn has demonstrated that **in the presence of alkalies glucose undergoes a stereochemic intramolecular change, becoming fructose, and then mannose;** with the result that the reduction of copper decreases and the plane of light is rotated toward the left, or is not rotated at all. They find that in soda solutions of the alkalinity of the blood glucose apparently disappears in considerable amounts, but this is probably a result of this stereochemic change, and not of the actual decomposition of the sugar. The authors do not claim that there is no such thing as fermentative glycolysis in human blood, but insist that there has never been any satisfactory demonstration of its existence or of the degree of its activity under normal or abnormal circumstances.

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[1] Deut. med. Woch., May 29 and June 5, 1902.

[2] Deut. med. Woch., Jan. 2, 1902.

R. Lépine,[1] in reply to Bendix and Bickel, states that he knew of Lobry de Bruyn's work, and that in his recent work he has excluded the errors due to this change in the sugar, by keeping the temperature below 60° C. (140° F.), and by adding slightly acid methyl alcohol. He has also avoided bacterial contamination. He states that while observing these regulations he found that there was a decrease in the dextrorotation and in the reducing power of the blood from a dog, after exposing it to body-temperature for a period of time; and that the fermentable sugar disappeared completely, or almost completely; these changes were not observed in the blood of a dog whose pancreas had been removed. The author also states that in his recent writings he **does not attribute glycolysis in the blood to a distinctly enzymotic process,** and that he has described the function of the pancreas in carbohydrate metabolism as being probably the destruction of substances which hinder glycolysis.

E. Bendix and A. Bickel[2] reply further to Lépine's article, and more particularly note that even though the latter did use acidified alcohol in his work, this is by no means satisfactory evidence that he used a good method in determining the blood-sugar. They say that sodium bicarbonate has the same action upon dextrose as soda itself, and give figures to show the apparent reduction that occurs in the dextrose. Pure hemoglobin solutions or small quantities of human blood, acting upon a neutral sugar solution at body-temperature, produce no reduction in the sugar.

Symptomatology.—Joslin[3] reports some **studies of metabolism** in a case of diabetes in a boy of 15, whose symptoms appeared in August, 1897. He notes that the **ferric-chlorid reaction showed very striking variations,** and particularly that the diacetic-acid excretion was greatly reduced during periods of freer diet; he attributes this to the more satisfactory oxidation of the acids, in the presence of carbohydrates, sufficient of the latter being oxidized, when they were given in large amounts, to cause satisfactory oxidation of oxybutyric and diacetic acid. The amount of acid excreted varied directly with the amount of urine, **diuresis evidently favoring the excretion.** This is to be expected, since betaoxybutyric acid is excreted only in dilute solution. This may explain the occasional recovery from diabetic coma when diuretics and normal salt infusions are used. Albumin was present in traces only, and casts were absent. The urine remained acid throughout, in spite of the administration of 140 grams of soda a day; while, in a normal person, 10 to 15 grams suffice to render the urine neutral or alkaline. The author found that the Squibbs method of estimating the urea was wholly unreliable in the presence of large amounts of acetone, ammonia, and oxybutyric acid. At the period of coma, acetone was found present in large quantities.

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[1] Deut. med. Woch., Jan 23, 1902.

[2] Deut. med. Woch., Mar. 6, 1902.

[3] Jour. of Med. Research, Nov., 1901.

A. C. Cotton[1] reports the case of a **girl of 6 years** and 3 months who **developed diabetes after an attack of measles,** the distinctive symptoms appearing in about 4 months after the measles. The family history showed no diabetes, gout, tuberculosis, or syphilis. The patient died, about 11 months after the attack of measles, in dyspneic coma. The author refers to Stern's statistics concerning diabetes in New York city; and states that in Chicago, for the four years 1897 to 1900, inclusive, 418 deaths from diabetes occurred, of which 3 were in children under 1 year and 15 in children under 10. These, including Stern's statistics, show, **in 2285 deaths from diabetes mellitus, only 0.31 % in children under 10 years old.** Diabetes is certainly at times hereditary, and Cotton considers that **prophylactic measures may well be employed,** in case there is a history of gout, diabetes, tuberculosis, or syphilis in the immediate ancestry; these should include the restriction of saccharine foods, the avoidance of mental or physical strain or shock, etc. If incontinence of urine or muscular debility, irritability, or emaciation appears, the prophylactic measures should be made more rigid. The mortality is high in children, and the diet needs to be very carefully regulated.

G. Hodge[2] reports a case of **diabetes in a boy of 16,** in which the **first signs appeared 5½ days before death.** Coma ended the patient's life. A careful investigation of the history failed to show any signs of the disease earlier than the time mentioned. A second case reported occurred in a girl of 7. This case was apparently of about 3 months' duration. The third patient was a girl of 5 years and 4 months. She had been passing urine too frequently and had seemed tired for some days before she was first seen. Three days after being seen she exhibited signs of oncoming coma, and 2 days later died in coma.

Marchi[3] reports a case of **diabetes in a child of 12,** some of the noteworthy facts concerning which were that the urine, which had amounted to 6 or 7 liters a day, was, by strict diet and opium and tannin medication, reduced to about 1 liter; while the excretion of sugar was reduced only from 10 % to about 7 %. The excessive hunger and thirst disappeared. There was a free excretion of diacetic acid, even with a good general condition and an increase in weight.

Rondet[4] describes a case of **diabetes of acute course in a boy.** Coma came on, and an effort was made to give the patient an intravenous injection of sodium bicarbonate; but the cannula could not be engaged in the vein, and subcutaneous injections were made in three regions. All told, about **600 grains of sodium bicarbonate were administered.** Consciousness soon partially returned, and the next day the patient was decidedly improved. Coma came on again, however, and the injections were repeated. Consciousness again returned for about 24 hours, and then deep coma appeared and the boy soon died. In the discussion

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[1] Jour. Am. Med. Assoc., Sept. 7, 1901.

[2] Canad. Pract. and Rev., July, 1901.

[3] Gaz. med. di Torino, 1901, No. 26.

[4] Lyon méd., July 7, 1901.

Lépine objected to the use of salines hypodermically, because of the danger of producing abscesses in diabetes.

E. Schwarz[1] exhibited to the Vienna Medical Society **a boy of 14** who, a year before, had been taken suddenly sick with **coincident myelogenous leukemia and diabetes mellitus,** the blood at that time showing over 300,000 leukocytes of a typical myelemic character, the spleen and liver being enlarged, and the lymph-glands being swollen. The urine contained 5 % of sugar. There was also an affection of the labyrinth and ascites. At one time 4 liters of ascitic fluid, containing 3 % of sugar, were withdrawn. There was no family history of diabetes.

J. B. Betts[2] describes the case of a man of 40 who had had **chronic melancholia** for about a year. Shortly before admission he showed emaciation and weakness, and the **urine contained 8 % of sugar.** The autopsy showed a normal number of the islands of Langerhans; but many of these were not clearly defined, and some were surrounded by a marked connective-tissue capsule. There was some atrophy in the head of the pancreas; in this region the islands were scarce, and there was some fibrosis about them. There was also old disease of the heart and liver and marked arterial change.

G. E. Herman[3] reports a case of **diabetes in pregnancy** in a woman of 30. The disease probably arose during the pregnancy. The woman died soon after the birth of a premature, dead child. This termination is comparatively rare, because diabetes usually interferes with the occurrence of pregnancy. The prognosis is usually better in those cases in which the disease arises during pregnancy than in the instances in which women already diabetic become pregnant. Pregnancy, in such cases, usually ends in the death of the fetus. Hydramnios is often present. Collapse and coma have repeatedly occurred after labor.

Zaudy[4] reports an interesting case of diabetes in a man of 63, the disease having lasted more than 2 years at the time of death. From September 14th to October 23d it was known that the urine contained large quantities of acetone and diacetic acid. It also contained albumin, had a high specific gravity, and showed large amounts of sugar. The first examination after October 23d was on November 1st, when careful testing showed the **absence of acetone, diacetic acid, sugar, and albumin,** and normal specific gravity, these conditions persisting until the time of death, which occurred on November 4th. There was no question of lack of carbohydrates in the food, or of inanition; for the patient was eating quantities of food, including large amounts of carbohydrates. The author thinks that the acid intoxication was due to the rapid destruction of body-fats, and that the disappearance of the signs of intoxication was due to either a cessation of the breakdown of body-fats or the patient's **reacquiring the power of using carbohydrates.** The latter is, Zaudy believes, the more probable; since an entire disappearance of fats from the tissues practically never takes place.

[1] Centralbl. f. innere Med., No. 12, 1902, p. 305.

[2] Albany Med. Ann., Sept., 1901.

[3] Edinb. Med. Jour., Feb., 1902

[4] Deut. med. Woch., June 5, 1902.

It is unreasonable to think that the man had lost the energy to destroy fats, because he was taking large amounts of food and was in fair condition until just before the time of his death. [The condition is very suggestive, because of the opinion that has been repeatedly expressed, that the catabolism of carbohydrates has an influence upon the normal catabolism of fats and proteids; and that, in a derangement of the former, there occurs a derangement of the latter, which results in the intoxication. In this case the disappearance of acidosis might very readily be due to the reacquisition of some power of using carbohydrates, and to its favorable influence upon the course of fat catabolism.]

K. Rössler[1] has **examined the extract of the feces** from numerous diabetics by the polarimeter, to determine the quantity of sugar present, and has found that on an average there is 0.0684 %; while the stools in normal persons contain no glucose. The amount in the stool in diabetics can be increased by giving glucose or lactose; while maltose and levulose cause no increase, but rather a decrease. Pentoses were not found in the stool.

Complications.—L. Oeder[2] refers to the fact that von Noorden and other writers on diabetes state that **patients with diabetes who acquire pulmonary tuberculosis** will probably not live for a long time, and that there is practically no hope of improvement. Oeder reports a case to show that this statement is not necessarily true. The man whose case he describes was seen in 1895, and was found at that time to have pronounced infiltration of the whole left apex, and signs of less marked infiltration down to the left base. The sputum contained large numbers of tubercle bacilli. The man also had a colonic fistula which was the result of several earlier operations for perityphlitic abscess. An examination of the urine undertaken merely in a routine way showed the presence of 7.5 % of sugar with a daily quantity of 1.25 liters of urine. The man apparently did not present the other characteristic symptoms of diabetes, except that he had decidedly lost weight. His case seemed to be very unfavorable, but he improved under treatment and disappeared from observation. When seen in the early part of this year [1902], it was learned that he had been growing very much better; that for a long time he had not been under treatment, and had been throughout that time eating whatever he pleased, with the exception of sugar itself. Examination showed that there were but slight signs of infiltration of the left lung (some impairment of resonance and weakness of the breath-sounds without any râles). The urine contained 3.3 % of sugar in 1.5 liters of daily urine, but the man's general condition was thoroughly good; he felt well, and was wholly capable of work.

Peacocke[3] reports a case of diabetes in which **hemichorea of the left side** developed, the condition persisting for 9 days. The man afterward developed an acute attack of right-sided parotitis. The case occurred in a man of 64, and ended fatally. There was no autopsy. The author was unable to find records of these complications in diabetes.

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[1] Zeitschr. f. Heilk., 1901, Hefte 8 u. 9.

[2] Deut. med. Woch., Oct. 3, 1901.

[3] Dublin Jour. Med. Sci., Jan., 1902.

Ellinger and Seelig[1] discuss the **influence of injury of the kidneys upon the course of pancreatic diabetes** in dogs, reporting their results with 15 animals. The injection of cantharides caused a diminution in the output of sugar, the drop being as great as from 7.6 % down to 2 %; and the dextrose-nitrogen ratio falling from 2.5 to 1. The influence of cantharides was only transient. In animals with peritonitis a diminution in the sugar-output occurred; and these animals showed kidney lesions. The authors likewise show that the dogs had hyperglycemia. They also refer to the work of Schaffer, who demonstrated that a unilateral kidney lesion causes a reduction in the sugar-excretion from this kidney. Ellinger and Seelig believe that the **increased tolerance for carbohydrates exhibited in cachexia is largely due to the occurrence of kidney lesions** in such states. They believe that much **attention should be paid to the examination of the urine for albumin and casts,** in such conditions; and that the sugar-content of the blood should be carefully determined. They refer to the occasional occurrence of large numbers of casts just before the onset of coma (Külz), and think that a retention of excessive amounts of sugar and other substances in the blood may indicate that imperfect kidney-action is of importance in the production of coma.

E. Nebelthau[2] reports some experiments concerning the **influence of fever with infection upon diabetes mellitus,** his method of experimentation being extirpation of the pancreas (the double operation), and infection of the animals with diphtheria bacilli, tetanus bacilli, streptococci, and tubercle bacilli, and intoxication with diphtheria and tetanus toxins. He also made observations of the sugar-excretion in cases of spontaneous infection occurring after the operation. The decision he reaches is that **infection and fever have no constant or definite effect upon diabetic glycosuria,** for the sugar-excretion was not notably influenced. In one case, however, in which there was a slowly progressing infection with tuberculosis, he found that the sugar-excretion diminished and finally disappeared before the dog's death. This animal had become very greatly emaciated, and practically all its fat had disappeared. The author decides from this that the organism may, under the influence of a chronic infection like tuberculosis, **reacquire the power of using carbohydrates,** if the fat has become so reduced in amount that the demands of the system for energy-producing material cannot be supplied by fat, and must, therefore, be met by carbohydrates, if at all. [It is, however, rather more probable that the organism, under these circumstances, loses still further its power of carbohydrate metabolism—that is, that it loses the ability to produce carbohydrates from proteids, and that, therefore, the excretion of sugar either becomes reduced or disappears for this reason.]

L. More[3] discusses the **influence of febrile diseases upon the glycosuria of diabetes,** and reports 6 cases of interest in this connection. He notes that with the exception of the fourth case they all belonged to the milder kind of diabetes before the febrile attacks, and that with the exception of the fourth and fifth cases the febrile attacks were all of mild character. Cases differ decidedly in their reaction to the febrile infections. In the second and third cases the slight fever caused decrease in tolerance for carbohydrates, but this disappeared shortly after the fever subsided. In the others a very striking effect was observed. Subsequent to the attacks in the first and sixth cases a long period passed before the patients reacquired the power that they had previously had of assimilating carbohydrates; in the fifth case the patient never reacquired the previous tolerance; and the fourth case, which had previously been an unfavorable one, showed after the febrile disease an increasing intolerance for carbohydrates and rapidly went on to a fatal issue. He therefore **divides the cases of diabetes, in relation to febrile effects,** into 2 classes: those in which the glycosuria is decreased, and those in which it is increased; and the last class can be subdivided into those in which there is a merely temporary increase during the fever, and those in which there is a decided subsequent action, and persistent increase in the severity of the diabetes. Fever acts in diabetes apparently by decreasing the functions of those organs which are active in the assimilation of sugar. There are two distinct elements of an infectious disease to be considered in relation to diabetes; *i. e.*, the elevation of temperature and the intoxication. **Fever increases the glycosuria and the hyperglycemia; intoxication often causes decrease in the blood-sugar.** The influence of an infectious disease is dependent upon these two factors: If the fever has the chief influence, the glycosuria will increase and the assimilation of sugar will decrease. If the infection has the chief influence, the contrary condition will be observed, temporarily at least. It may be that certain microorganisms have a specific action while others do not.

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[1] Verhandlung. des Congress. f. innere Med., 1901.

[2] Arch. f. exper. Path. u. Pharm., Bd. XLVI, Hefte 5 u. 6.

[3] Zeitschr. f. klin. Med., Bd. XLII, Hefte 5 u. 6.

R. Waldvogel and J. Hagenberg[1] contribute some **experimental investigations concerning alimentary acetonuria.** Fats are now generally thought to furnish the chief amount of acetone and of diacetic and oxybutyric acids. They therefore administered to a series of patients a constant diet, and occasionally added an excessive amount of fat—from 50 to 150 grams of butter or oil. They found that they could in this way, in almost all instances, largely increase the amount of acetone excreted in the urine. They therefore decided that **acetone is certainly a product of fats;** that it appears in abnormal amounts in urine when the demand for energy must be met by breaking-down of body-fats; and that it also **appears when there are large amounts of fatty substances in the intestine.** The latter is the case when there are digestive disturbances, or when large amounts of fats are taken in the food. They therefore divide acetonuria as follows: First, that resulting from the breaking-down of body-fats in metabolism; second, that due to the presence of fatty substances in the intestines, this second division having the subdivisions (*a*) enterogenous, due to an intestinal disturbance, and (*b*) alimentary enterogenous, due to the administration of large amounts of fats. They have also made control determinations of acetone by Waldvogel's method, and the Messinger-Huppert method, and state that the results with the former corresponded closely with those given by the latter. They therefore think that criticisms of Waldvogel's method are not justified, and that this method is quite as good as the other mentioned, and is decidedly simpler.

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[1] Zeitschr. f. klin. Med., Bd. XLII, Hefte 5 u. 6.

H. Lüthje[1] reports some further studies of the metabolism of a diabetic subject whose case has previously been reported. The patient was admitted into the hospital in coma. The use of soda solution intravenously and by the mouth overcame the coma; the author considers that the bicarbonate of soda is much better than the carbonate. The **coma did not cause a decrease in the sugar-excretion.** The ammonia excretion was high during this time. Lüthje particularly directs attention to the fact that he has **compared the excretion of sugar when casein was used with that when egg-albumen was employed.** It was decidedly increased by the use of casein; hence, the author believes that it is definitely shown that **sugar is split off from casein.** He is doubtful about the formation of sugar from fat, although he found the nitrogen-dextrose ratio very high; he believes that there is no use in discussing the question of the formation of sugar from fat, so long as the maximal sugar-production from albumin is not known. In his animal experiments he attempted to determine this ratio more definitely by removing the duodenum with the pancreas. The ratio of nitrogen to dextrose did not pass 1:2.8; but the experiment was unsatisfactory, because of the rapid death of the animal. **Absorption of the food in the case reported was good** up to the time of death. The excretion of diacetic and oxybutyric acids was high. The author **believes that the acid-excretion in diabetes is due to a special metabolic disturbance.** It is notable that during 3 days, when the carbohydrate of the food was chiefly milk-sugar, the patient **excreted both glucose and milk-sugar** with the urine.

K. Domanky and G. Reimann[2] report **3 cases of diabetic coma in which they found Külz's casts.** Microscopic examination of the kidneys showed a high grade of parenchymatous change. They believe that the appearance of the casts in the urine is of great value as a sign of the onset of coma, and is important in prophylaxis. The condition of the urine in diabetics first causes hypertrophy of the kidney, and this is likely to be followed by actual damage to the kidney-parenchyma; and they believe that in many cases of diabetes the damage to the kidney-elements results in retention of the constituents of the urine and the consequent appearance of uremic symptoms.

Diagnosis.—E. Reali[3] discusses the **quantitative determination of the carbohydrates in the urine.** He describes a method by means of which he thinks one may determine the amount of preformed reducing-sugar in the urine; also the amount of sugar present which does not reduce copper. He precipitated the urine with neutral lead acetate and ammonia, and dissolved the precipitate in a soda solution which contained Rochelle salts. This solution contained all the reducing-sugar. If the urine causes no reduction in this way, the precipitate may be boiled gently with 100 cc. of 25 % HCl for a quarter of an hour, neutralized with soda, and then the reduction tested. This will give a positive result, and thus the total amount of carbohydrate in the urine may be determined. [It is probable that the glycuronic acid in glycuronic compounds produces much of the second reduction in many cases, and this error certainly makes the method inexact.]

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[1] Zeitschr. f. klin. Med., Bd. XLIII, p. 225.
[2] Zeitschr. f. Heilk., 1901, Hefte 8 u. 9.
[3] Rivista clin. e therap., No. 5, 1901.

Allard,[1] in a discussion of diaceturia, notes his results with **Arnold's and Lipliawsky's reactions.** Arnold's reaction with paramidoacetophenon, he states, gives a positive result with a quantity of diacetic acid equal to about 0.01 %. Lipliawsky's reaction will show diacetic acid in a solution of 0.004 %. The latter reaction is carried out by using a 1 % solution of paramidoacetophenon and a 1 % solution of sodium nitrite; 3 cc. of the first and 6 cc. of the second solution are added to the same quantity of urine; a drop of ammonia is added; the mixture is energetically shaken; from 10 drops to 2 cc. of this is then taken; 15 to 20 cc. of strong HCl, 3 cc. of chloroform, and 2 to 4 drops of ferric chlorid solution are added; the test-tube is then corked, and repeatedly but very gently inverted. If diacetic acid is present, the chloroform takes a characteristic violet color. Concentrated ammonia and HCl, of a specific gravity of 1.19, must be used. Acetone solutions of more than 1 % strength give both Arnold's and Lipliawsky's reactions. Gerhardt's method remains the simpler and more useful one for clinical purposes.

C. A. Herter[2] considers that a simplified method of **balancing the acids and bases in the urine** provides a satisfactory way of **determining the degree of acid intoxication.** He particularly insists that the determination of the ammonia of the urine is not sufficient to demonstrate the presence or absence of decided acid intoxication, as he finds that the acids are often excreted in combination with the fixed bases, when the ammonia shows little or no increase. As much as 25 or 30 grams of oxybutyric acid may be excreted and yet the patient still seem in good condition; but under such circumstances coma is likely to appear soon. In rare instances the acid-excretion spontaneously decreases. There is no direct relation between the amount of sugar-excretion and that of acid-excretion; and in order to determine the actual clinical condition of the case of diabetes, with especial regard to the possible occurrence of coma, it is **essential to determine the acid-excretion.** Sometimes the acid-excretion may be favorably influenced by withdrawing carbohydrates; in other cases this is not true. If a considerable amount of oxybutyric acid is formed with a carbohydrate-free diet, the prognosis is bad. At times, the addition of carbohydrates to the diet has a favorable influence upon the acidosis.

W. A. Boekelman and J. Bouma[3] report a new method for the **determination of the amount of betaoxybutyric acid** in the urine. The difficulties in the ordinary method are well known. The authors proceed as follows: To 25 cc. of urine in a flask they add the same quantity of 12 % sodium hydrate solution and of benzoyl chlorid; they then cork the flask and shake it energetically, while holding it under a stream of cold water. The precipitate is allowed to settle, the overlying liquid is introduced into a polarimeter, and the betaoxybutyric acid is determined by the reading.

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[1] Berl. klin. Woch., No. 39, 1901.

[2] Jour. Exper. Med., vol. v, p. 617

[3] Feestbundel, Dr. S. Talma, 1901, p. 157.

Treatment.—Talma,[1] in a discussion of the **nourishment of diabetics,** states that, as a rule, the **daily total nitrogen of the urine should be kept at about 16 grams;** but that, particularly in gouty subjects, this figure is sometimes too high. The nitrogen in the latter cases must be reduced; and then one must take the utmost care to see that, while the nitrogen-excretion is kept down, the patient is not losing tissue. If the patients complain markedly of hunger, and of a physical and psychic loss of energy, this is often an indication that they are getting too little nitrogenous food; and these symptoms will often disappear after increasing the protein. The increase in the glycosuria after the prolonged use of protein food is, at times, in the author's belief, due to disease of the kidneys, and results from excessive and prolonged nitrogen-excretion. It is, as yet, unknown whether this renal increase in glycosuria is more likely to occur in those who are subjects of gout or of the gouty diathesis; **actual gout and diabetes are not often combined** A high nitrogen-content of the urine is not often harmful, and becomes evidently harmful only when it causes albuminuria or oxaluria. If dyspeptic symptoms appear, a part or, at times, all of the food should be temporarily excluded. When fats are being used in large amounts, the function of the gastrointestinal tract should be very carefully watched; because, under these circumstances, the **fats are particularly likely to increase the acid intoxication.** If dyspeptic symptoms are absent, the main fact to be considered is the body-weight.

H. Camerer, Jr.,[2] has made a study of various **breads recommended for diabetics.** Of 10 breads, all of European make, he found that none fulfilled the requirements which a diabetic bread should exhibit. The amount of carbohydrate in them was not below 40 %. When kept for a long time, these breads, and all others, lose water; consequently, the relative percentage of carbohydrate increases. As a rule, one should reckon that the **diabetic breads contain as much as 30 % of carbohydrate.** At the same time, they are less pleasant to take than ordinary breads and are more expensive. The author recommends the preparation at home of a rye bread in which fat, as well as tropon or plasmon, is kneaded. This preparation can be made to contain only about 25 % of carbohydrate, and is said to be taken readily when fresh.

Jacobsohn[3] demonstrated to the Berlin Medical Society a preparation of **"robora" diabetic bread,** which, he claims, meets all the indications that a diabetic bread should meet. It can be kept for a long time, satisfies hunger, and contains large amounts of albumin and relatively large amounts of fat.

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[1] Therapie der Gegenwart, Sept., 1901.

[2] Zeitschr. f. diät. u. physikal. Therap., Bd. v, Hft. 3.

[3] Centralbl. f. innere Med., April 19, 1902, p. 410.

A. Mossé [1] gives a somewhat extensive discussion of his use of **potatoes in the treatment of diabetes** and its complications. He claims to have produced a very marked amelioràtion of the general condition and an improvement in the glycosuria in this way. He has some doubt as to whether potatoes will always give satisfactory results in diabetes, and thinks that probably one method of treatment is not applicable to all cases of the disease; but he is, nevertheless, enthusiastic. In only 1 of 20 cases was a satisfactory result not obtained, and this was in an arthritic person, in whom the diabetes was mild.

E. Allard, [2] after a study of the influence of Mergentheim Karl Spring water upon obesity and diabetes mellitus, reaches the conclusion that the use of this water causes a reduction in fat without a loss of nitrogenous tissue, and also seems to reduce the sugar-excretion in diabetes mellitus.

R. T. Williamson, [3] in a general discussion of the treatment of diabetes, states that he has **used heroin,** and has found that it has not so much of a tendency to produce constipation as opium and other derivatives have. In several mild cases it **caused the sugar-excretion to cease.** In two cases the drug produced severe vomiting, probably as the result of some impurity or of decomposition. He has had better results with sodium salicylate than with any other drug; and in cases in which the diet was kept exactly the same, he thinks that he has demonstrated very positively that it has an important effect in restricting the sugar-excretion and, at times, in causing it to cease. He has also used aspirin in a number of cases with good results.

L. Schwarz, [4] in discussing the **treatment of diabetic coma,** refers to the fact that the use of carbohydrate acids may reduce the acetone excretion. He was led by this to use **gluconic acid** in a case of coma; 70 grams was given in a half-liter of water, neutralized by sodium bicarbonate. The author also gave large doses of sodium bicarbonate. The urine remained acid, but the dyspnea and stupor disappeared during the day, and the next morning there was no sign of coma. Large doses of alkali were continued; but, after 3 weeks, coma appeared anew. Five hours afterward, 50 grams of **sodium gluconate** was given, with rapid improvement. Two weeks later, while the patient was comparatively well, there was a third attack of coma. Large doses of soda were given, but gluconic acid could not be used. The patient died in this attack. Schwarz is inclined to attribute the earlier results to the gluconic acid and its effect in reducing the amount of acids produced. However, one case is, of course, not sufficient to establish his point.

DIABETES INSIPIDUS.

K. v. Alfthan [5] estimated the **amount of animal gum in the urine** of 2 cases of diabetes insipidus, and found it rather decreased than increased, contrary to the conditions in diabetes mellitus.

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[1] Bull. de l'Acad. de Méd., Dec., 1901.

[2] Zeitschr. f. klin. Med., Bd. XLV, Hefte 3 u. 4.

[3] Dominion Med. Monthly, Dec., 1901.

[4] Berl. med. Woch., Nos. 30 and 31, 1901.

[5] Berl. klin. Woch., Feb. 24, 1902.

P. Kuhn[1] reports the case of a woman of 58 with sarcoma of the breast, who exhibited **diabetes insipidus** that had persisted for a year and a half. Eight days before death **glycosuria appeared, and persisted** until the end. No explanation of the glycosuria was found postmortem.

M. v. Zeissl[2] reports a case of diabetes insipidus in which there was a syphilitic history, and in which **anti-syphilitic treatment caused a marked improvement** of the general health and a decrease in the polyuria.

GOUT.

Etiology and Pathology.—A. Schittenhelm[3] contributes a study concerning **the behavior of adenin and guanin** in the animal organism, following the work of Minkowski, who had shown that adenin was very poisonous to dogs. Schittenhelm makes the interesting statement that when administered to rabbits, even in large doses (as much as 13 grams in 10 days), **no symptoms of poisoning were produced.** The **urine, however, contained ammonium urate** (?) in large quantity; although the total amount of uric acid was not increased. About 25 % of the adenin administered was recovered, as such, in the urine. The postmortem examination showed no changes, except the deposition of a large amount of crystals of what seemed to be ammonium urate. Evidently, the **effect of adenin is different in different species;** although the effect upon the kidneys was the same, except that rabbits required much larger doses than dogs. The effect of guanin has been studied by several other authors, and the results all showed that there was no increase in the uric acid, or purin bases; except in the case of human subjects, when there was a slight increase in the amount of uric acid. Schittenhelm found, in one rabbit, that, after administering *in toto* 5.3 grams of guanin sulfate, there were no pathologic changes discoverable at postmortem, and that there was no increase in the uric acid or bases.

Vogt[4] compared the **nitrogen and phosphorus metabolism** of a patient with gout with the same conditions in a normal man. In the first period the diet contained 14.58 grams of nitrogen and 2.621 of P_2O_5. During this period the healthy man lost nitrogen, and the gouty subject retained it; both lost P_2O_5. In the second period 175 grams of pancreas was added to the diet, which increased the daily intake to 19.31 grams of nitrogen and 4.861 grams of P_2O_5. During this period the healthy man retained nitrogen, and the gouty subject retained even more than before; both retained P_2O_5, the gouty subject more than the healthy subject. The diet of the third period was that of the first. During this period the healthy subject lost over 2 grams of nitrogen per day; the gouty subject lost 0.33 gram. The healthy subject lost over a gram of P_2O_5; the gouty subject lost about half a gram. It is notable that, while the normal man lost nitrogen in the first period, because the intake was too small, the **gouty subject showed nitrogen-retention constantly,** even though he had a slight attack of gout during the period of investigation. In the third period, although the nitrogen seemed about at an equilibrium, there was a **noteworthy loss of P_2O_5.** The author concludes that the nuclein is broken up and the P_2O_5 is at once excreted, the nitrogen remaining behind. The **question is whether the nitrogen retained is albumin-nitrogen or purin-nitrogen.** This question, he thinks, is answered by the study of the uric acid excretion. In the period in which all the P_2O_5 was being excreted, the uric-acid excretion was decidedly less in the gouty subject than in the healthy man. The author thinks that this indicates that the nuclein which is absorbed is normally broken up, and that the phosphorus contained in it is at once excreted, while the **excretion of uric acid is imperfect.** This may be due to imperfect oxidation, to abnormalities in the splitting-up of the nucleinic acid, or to abnormalities in the synthesis of uric acid. The excretion of nitrogen and uric acid in the gouty subject seemed to be relatively less than the excretion of P_2O_5; and this, at any rate, shows an **abnormality in the metabolism of the nucleins,** although Vogt considers it doubtful whether this is the sole cause of the nitrogen-retention in gout.

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[1] Münch. med. Woch., No. 3, 1902.

[2] Wien. med. Presse, Aug. 11, 1901.

[3] Arch. f. exp. Path. u. Pharm., Bd. XLVII, Hefte 5 u. 6.

[4] Deut. Arch. f. klin. Med., Bd. LXXI, Hft. 1.

E. Bendix[1] refers to the fact that the joints of gouty subjects not infrequently become exposed by the breaking-down of the tissues about uratic deposits, and yet that these joints rarely suppurate. This has led to the **statement that uric acid or urates have an antiseptic action.** He investigated this possibility by adding an excess of uric acid and sodium urate to cultures of colon bacillus, streptococcus, staphylococcus, and tubercle bacillus, and found that there was **absolutely no influence to be observed upon the growth of the microorganisms.** The infrequent occurrence of tuberculosis in gouty subjects is, he believes, attributable to the general constitution of such persons, rather than to excess of uric acid; and the absence of suppuration in gouty joints, or the absence of general sepsis in cases in which gouty joints do suppurate, is attributable to the marked thickening of the tissues about the joints, and particularly to the dense deposits of urates and other salts, which prevent the invasion of bacteria and the absorption of their products.

G. Klemperer[2] some years ago drew attention to the fact that the blood of gouty subjects, even during an attack, is still capable of causing solution of uric acid. He decides from this that substances such as piperazin, which have the property of dissolving uric acid, are not likely to be valuable in the treatment of gout. He also believes that those substances which apparently cause a decrease in the production of uric acid are probably of little value in this disease. It has been determined that, under normal circumstances, the tissues have the property of destroying uric acid; that is, they produce urolysis. The author has **tested the urolytic powers of the blood of gouty subjects,** and found them similar to those of normal persons; hence, the excessive amount of uric acid in the blood of gouty subjects is not due to imperfect destruction of the uric acid.

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[1] Zeitschr. f. klin. Med., Bd. IV, Hft. 1 u. 2.
[2] Therap. der Gegenwart, No. 8, 1901.

L. Zoja[1] reports that the **fluid withdrawn from the knee-joint** in a case of acute rheumatic polyarthritis **deposited uric acid crystals** after standing. One possible explanation of this is the fact that the patient was also the subject of lead-poisoning.

Treatment.—G. Klemperer[2] discusses the question as to **whether fish is more or less useful than meat in the uric acid diathesis** and in nephritis. Patients with kidney calculi should take either meat or fish in small amounts, together with large quantities of fluid and much vegetable nourishment. There is no definite knowledge whether fish or meat is the better in gout; but it is certain that excessive amounts of animal food are harmful, and that animal food should be eaten with relatively large quantities of vegetables. **Fish is certainly borne as well as meat by gouty subjects.** Klemperer investigated the influence of fish, as compared with meat, upon albuminuria in nephritis, and found that the albuminuria was less when fish was taken than when meat was given, but decidedly less still when milk was substituted for the fish or meat. These results, however, were obtained in only 2 cases, and may have been accidental.

W. Bergmann[3] refers to the fact that there have been **attempts to control the deposition of urates in the urine** in uric acid calculus, by administering calcium salts; the reason being that uratic deposits are largely due to the increased acidity of the urine, which is produced, to a great extent, by the phosphates. It was thought that the administration of calcium would cause a large portion of the phosphates to be excreted through the intestine as calcium salts. The author injected phosphoric acid subcutaneously in a dog, and found that practically none of this substance was excreted through the intestine. He then coincidently administered calcium and phosphoric acid, and found that the phosphoric acid was still excreted entirely through the urine. In herbivorous animals he found that practically all of the phosphoric acid is normally excreted through the intestine. He also found that glycerin-phosphoric acid injections demonstrate that organic phosphorous combinations are excreted through the urine in the dog, through the intestine in the sheep; in both cases as inorganic salts. His results indicate that **calcium is valueless in controlling uratic deposits** [a result which corresponds with the report of Ury, mentioned in this volume, which apparently demonstrates that in human subjects very little calcium is excreted through the intestine, most of the calcium produced in metabolism passing out through the urine].

H. Ulrici[4] gives a **study of the influence of certain pharmacologic preparations upon the excretion of uric acid.** He investigated the influence of benzoic, gallic, quinic, tannic, and sulfuric acid upon metabolism and uric acid excretion, the diet being kept constant, the nitrogen and phosphorus being determined in both the intake and the outgo, and the uric acid excretion being regularly determined. The **influence of**

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[1] Gaz. med. di Torino, 1901, No. 34.

[2] Therap. der Gegenwart, Sept., 1901.

[3] Arch. f. exper. Path. u. Pharm., Bd. XLVII, Hft. 1 u. 2.

[4] Arch. f. exper. Path. u. Pharm., Bd. XLVI, Hft. 5 u. 6.

benzoic and gallic acid was very decided; both produced a marked reduction in the excretion of uric acid. Both of them also caused a very slight reduction in nitrogen metabolism. The author does not think it possible that the reduction of uric acid in these instances could have been attributed to a reduction in synthesis of uric acid from glycocoll; as the reduction was followed by an increase in the excretion; and, although this increase was not sufficient to offset the previous reduction, it was enough to indicate that there was retention of uric acid, rather than that the reduction was, in its entirety, due to reduced formation. **Quinic acid,** contrary to the results of Weiss, **had no discoverable influence.** Tannin, likewise, had no notable influence. **Salicylic acid,** on the contrary, **caused a very decided increase** in the uric acid excretion—an increase which could not be attributed merely to flushing of the tissues of the uric acid that was already formed; it was, he believes, a specific effect of salicylic acid upon metabolism. The **influence of salicylic acid was, therefore, directly contrary to that of gallic and benzoic acid.** The author also reports his results from coincident estimation of the **number of leukocytes and of uric acid elimination.** He insists that in such experiments one should not depend upon the absolute number of leukocytes, but upon their relative number, as compared with the red corpuscles. He finds, with other recent workers upon the same subject, that there is no definite relation to be observed between the uric acid and the number of the leukocytes after the administration of sodium salicylate; this drug, while producing an increase in uric acid, causes no notable change in the leukocytes.

De la Camp[1] has investigated the **influence of chinotropin** upon metabolism and the excretion of uric acid. He finds that it has no constant influence upon uric acid excretion, either in gouty subjects or in normal persons; but that it decidedly increases the hippuric-acid excretion. Uric acid seemed to be decreased when large amounts of nourishment that produces uric acid, such as thymus, were used, or when, as in leukemia, the organism was producing large quantities of uric acid. The author believes, however, that clinical results speak in favor of the use of this substance in gout; and it is harmless, even in large doses. He thinks that it is likely to be useful in uric-acid calculus formation, because formaldehyd is set free from the urotropin contained, and this dissolves uric acid.

G. Klemperer[2] states that **free carbonic acid in the urine** renders the solution of uric acid difficult, but makes the solution of urates easier. The effort, therefore, should be to secure a neutral or nearly neutral reaction of the urine, with a considerable content of carbonic acid. This is best accomplished by a mixed diet and the use of alkaline mineral water containing CO_2.

RHEUMATOID ARTHRITIS.

A. E. Garrod[3] opened a very interesting discussion on **chronic diseases of the joints** commonly included in the terms chronic rheumatism, osteoarthritis, and rheumatic gout. He particularly directs attention to the probability of our **confusing under the one name, rheumatoid arthritis, a number of conditions which actually have nothing in common.** As to the pathologic changes seen in the joints, he insists that it is open to question whether they are or are not merely the changes which occur in joints that are the seats of long-standing disease of various kinds; and that it is certainly doubtful whether the same changes are present in all the cases called arthritis deformans. He finds **at least two well-defined groups:** the first, the more acute variety of the disease, in which acute exacerbations occur, and give rise to spindle-shaped swelling, with effusion into the synovial cavities and decided muscular atrophy—this form often occurring in comparatively young persons, and not infrequently being associated with decided fever; the form of disease described by Still is probably identical with this variety. The second group of cases includes those in which the lesions tend to be decidedly symmetric, to attack the small joints, to occur later in life, to be associated with practically no constitutional disturbance and commonly with very little pain, and to produce almost nothing but disfigurement and limitation of movement—the enlargement of the joints in these cases being almost entirely the result of bony outgrowth. These two conditions are probably not merely stages in the same disease, but are distinct from the outset. There is still another type, which is probably also distinct; in this, articular swelling is comparatively slight, but there is marked crippling from muscular contraction and fixation of the joints. In one of these cases Garrod has actually found **bridges of cancellated bone between the denuded articular surfaces.** He discusses the probable relation of these conditions to acute rheumatism and to gout. They **cannot be looked upon as being simply acute rheumatism,** although it is possible that they occasionally develop on top of a rheumatic attack; but in most instances the symptoms are probably due purely to arthritis deformans from the outset. Endocarditis is certainly uncommon in arthritis deformans, and the nodules which occur in the latter disease are likely to be more persistent than those that occur in rheumatism. The **relation to gout is even more unsettled.** Garrod inclines to the view that there is no closer relation with gout than with acute rheumatism. The form of the disease first mentioned has **much to suggest an infective origin;** while in the nodular or osteoarthritic lesions of older people the origin is probably purely dystrophic. In the treatment the author especially insists upon the importance of care of the general health, particularly of the digestive tract; and especially notes the danger of overlooking unhealthy conditions of the mouth or gums, which may be important in producing the infection. These patients are very **frequently put upon too low a diet,** with the idea of a relationship to gout. Garrod also does not recommend the cutting-off of stimulants. Fresh air, tonics, and massage to the muscles, he strongly recommends. **Massage and passive movements of the joints themselves probably do more harm than good.** He particularly insists that the degree of good that can be accomplished is not usually appreciated.

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[1] Münch. med. Woch., No. 30, 1901.

[2] Zeitschr. f. diät. u. physikal. Therapie, Bd. v, Hft. 1, 1901.

[3] Brit. Med. Jour., Oct. 12, 1901.

W. Armstrong strongly insisted that the **nervous theory** of the disease is much more satisfactory than the bacterial; and believes that the nerve-nutrition of the joints is interfered with in many cases.

A. P. Luff is strongly inclined to the **bacterial theory** of the disease. He believes that the monarticular form is entirely distinct from the general form.

W. P. Herringham referred to the **difficulty in diagnosing some cases from acute rheumatism,** and especially mentioned the importance of the occurrence of heart lesions and of the absence of permanent joint lesions; while in arthritis deformans there are permanent joint lesions and no heart lesions. He firmly believes in the unity of the different conditions which there has recently been a tendency to separate into several varieties.

C. O. Hawthorne also believes that the different conditions cannot be classed separately, and can see **no distinctive symptoms of what is commonly called chronic rheumatism.** He inclines to the belief that it would be **better to call all chronic joint conditions merely chronic arthritis,** adding osteoarthritis or arthritis deformans if especial clinical conditions justify this. He had found that sodium salicylate often gives a great deal of relief from the pain and stiffness.

W. Calwell suggested that arthritis deformans **is not improbably a water-borne disease,** and insisted upon the lack of proper personal hygiene as an important element in its causation.

E. J. Cave, in a very comprehensive view of the subject, considered that these **joint lesions undoubtedly have a manifold causation,** and that we may most reasonably—excluding traumatism and senile degeneration, and those forms obviously associated with nerve lesions—attribute them to the **result of many infections;** and he thinks that there is probably no one specific clinical or bacterial condition which may be termed arthritis deformans. He referred to the fact that the pneumococcus may produce a lesion wholly similar to rheumatoid arthritis, and to the further fact that rectal ulceration may set up similar lesions. He described the case of a boy who had ulceration of the rectum; and who—after the first fortnight of his illness—exhibited swellings of the small joints, which gradually advanced to various other joints, the joint condition showing numerous subacute exacerbations, and being associated with extreme muscular atrophy and marked pigmentation of the skin. There was ulnar deflexion, and the boy ultimately presented the most typical appearance of rheumatoid arthritis. A colotomy was undertaken; the **rectal ulceration was cured; and with this there was the most striking amelioration of the joint condition.** Cave believes, then, that the various forms of arthritis may result from the same infection, and that the same form may result from various infections; and he doubts whether there is any specific infection that causes any special variety of chronic progressive arthritis. In the treatment of the condition he insists strongly that it is absolutely essential to recognize that in many of these cases, at any rate, **there is some local source of infection;** that we must look with the greatest care to the mouth, nose, ear, the accessory cavities, the alimentary tract and its diverticulums, the skin, the respiratory tract, the genital and urinary organs, etc.; and that if a source of infection can be removed, it will often aid greatly in arresting or in largely improving the condition. He considers that the **physical methods of cure are of great value.** Heat and cold, forced movements under anesthesia, the maintenance of the movement thus gained, massage, and electricity are extremely important.

S. West fully agrees with Cave in regard to the question. As an evidence of the importance of local infection, he described a case of slight chronic cystitis; **with the cure of the cystitis, the joint symptoms disappeared.**

E. M. Merrins,[1] in discussing the **nature of chronic rheumatism,** concludes that it is probable that we shall come to class chronic joint affections as: (1) traumatic, (2) due to bacterial infection, (3) the result of the action of toxins and other chemical irritants, (4) the outcome of nerve-degeneration.

Duer[2] states that **rheumatism and osteoarthritis, as well as gout, are rare in India,** even in the natives, who live in most unhealthful climatic and local conditions; and in the latter patients, severe fever and heart complications are unusual. These diseases seem to be comparatively rare and mild, even in Europeans. This observation may be of use in the climatic treatment of such patients.

L. Kast[3] has carried out a study of the condition of **the heart in chronic rheumatism and arthritis deformans.** In the 36,017 postmortem protocols of the Pathological Institute in Prague, there were 24 instances of chronic articular rheumatism or arthritis deformans. A study of these postmortem examinations and of the clinical histories of the cases led the author to the conclusion that, in relation to endocarditic changes, there is a **strict distinction to be made between those cases which are, from the first, chronic, and those beginning with acute polyarthritis.** In 17 cases with clear histories, 4 had an acute beginning, and 75 % of these had cardiopathies; 13 were chronic from the beginning, and 31.8 % of these showed cardiopathies. Those cases which are primarily chronic are extremely likely to be complicated with tuberculosis, Bright's disease, and chronic deforming endarteritis; and they are also likely to occur in persons of more advanced age. The cases with acute onset are likely to die of the cardiac disease. Those that are chronic from the beginning usually terminate in death by tuberculosis or Bright's disease.

H. Forrestier[4] reports 23 cases of **rheumatic spondylitis.** He divides them into (1) cases in which the whole spinal column is involved, in which there is pain of moderate severity, but in which resolution usually occurs; (2) cases in which the vertebral column is involved throughout, in which rigidity occurs early, and in which there is usually severe neuralgic pain and actual ankylosis; and (3) cases in which there is severe pain, with rigidity of the whole column, but recovery takes place. He **divides spondylitis into severe, ankylosing, and pseudoneuralgic.** The latter form is very likely not to be properly diagnosed. It is most commonly observed after chronic rheumatism. Hot baths and massage are useful in the treatment.

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[1] Med. Rec., Mar. 22, 1902.

[2] Brit. Med. Jour., Dec. 28, 1901.

[3] Prag. med. Woch., Nos. 41–44, 1901.

[4] Arch. gén. de méd., July and Aug., 1901.

H. S. Upson[1] discusses the use of **thiosinamin** in chronic joint affections, and describes several cases in which it was used with inconclusive results.

DISEASES OF THE THYROID GLAND.

Von Mikulicz and G. Reinbach[2] have made a study of the **relation between disease of the thyroid gland and exophthalmic goiter.** They studied the collateral symptoms appearing with simple, uncomplicated goiter in 117 cases. Of these, 48 showed absolutely no nervous, cardiac, or psychic collateral symptoms; in 8 there was slight increase in the rapidity of the pulse; in 14, very slight suggestion of exophthalmos; and in most of the 69 cases which showed collateral symptoms there were slight nervous symptoms—most commonly tremor of the hands and tongue, headache, sometimes subjective palpitation and vertigo, anginoid pains, restlessness, and irritability. The authors never observed any pronounced increase in the frequency of the pulse, marked exophthalmos, or decided nervous or psychic symptoms; and all the trophic disturbances which are frequently seen in exophthalmic goiter were entirely absent. They come to the conclusion that **exophthalmic goiter cannot be considered as essentially an excessive functionation of the thyroid** gland; but whether it be considered to be a neurosis or an autointoxication, **disturbances of the function of the thyroid undoubtedly play a prominent part.** They think, however, that the thyroid gland hypertrophies as a result of the same influences that cause the changes in other organs. The symptoms of thyroidism are, therefore, due to the changes in the thyroid gland, but the **changes in the gland are secondary.** The thyroid, in other words, acts as a multiplicator.

W. Edmunds[3] insists that, whether the anatomic disturbance in Graves's disease be chiefly in the nervous system primarily, or elsewhere, the **main disturbance is originally of a chemical nature.** Experiments were made upon 8 dogs; in 4 excising the thyroids and parathyroids at once, and in the other 4 excising the thyroids and parathyroids at two sittings. He then investigated the central nervous system, and found very marked changes in the Nissl bodies, similar to those found in acute toxic states, thus indicating that athyroidism is a form of acute poisoning severely affecting the central nervous system. The acute nervous symptoms can be produced by removal of the parathyroids alone, while they are not produced by excision of the thyroid alone.

[1] Amer. Med., Jan. 25, 1902.
[2] Mittheil. a. d. Grenzeg. der Med. u. Chir., Bd. VIII, Hft. 3.
[3] Brit. Med. Jour., Sept. 21, 1901.

Hence, the absence of the parathyroid secretion seems to be the cause of the acute symptoms; and, since it has been demonstrated that there is a large amount of iodin in the parathyroids, it is very probable that **iodin plays an important part in the prevention of these symptoms.** The symptoms of Graves's disease he considers to resemble those following removal of the parathyroids much more closely than those due to the removal of the thyroid itself, and he insists that the parathyroids should be carefully examined in autopsies on cases of Graves's disease. This affection may be due to partial aparathyroidism.

Mannini[1] considers that exophthalmic goiter is most commonly a **neurosis of the cervical sympathetic.** He notes the frequency of rudimentary forms of exophthalmic goiter in psychic disturbances, and discusses 2 cases which occurred in a hysteric and in a melancholic patient, in which there were partial signs of exophthalmic goiter. The signs of intoxication he considers to be secondary; those of neurosis, primary.

Biagi[2] has made an experimental study concerning the pathogenesis and the treatment of exophthalmic goiter. Extirpation of the sympathetic ganglia and coincident removal of the laryngeal nerves had no trophic influence upon the thyroid gland. The only result was the temporary dilation of the vessels; but no clinical signs of disturbance of the thyroid and no evidences of degeneration or regeneration appeared. The author's only conclusion is that **lesions of the sympathetic do not cause the thyroid changes.**

Campbell[3] reports in a woman of 27 a case of exophthalmic goiter which ran a **very rapid course,** death occurring after 2 months, apparently purely from the severity of the disease itself.

F. C. Eve[4] reports 2 cases of Graves's disease that were **complicated by insanity.** One patient died. The other recovered, her improvement dating from the time that venesection was carried out.

Kedzior and Zanietowski[5] describe the case of a girl of 18 who had **thyroid enlargement at the age of 14.** This was followed by tremor of the hands, weakness, exophthalmos, palpitation, a tendency to sweating, and dyspnea. The right lobe of the thyroid was resected, and the superior and inferior left thyroid arteries were ligated. The general condition of the patient was improved, and the rapidity of the pulse greatly decreased. Afterward, however, she grew much worse; epileptiform attacks developed, and death finally occurred. The autopsy showed croupous pneumonia, hypertrophy of the thymus, and dilation and hypertrophy of the heart. There were old and new hemorrhages scattered throughout the medulla, and the left restiform body was much smaller than the right. Microscopically, there were no further abnormalities in the central nervous system than these hemorrhages and a yellowish and waxy appearance of the restiform bodies. The left cerebellar tract and the nucleus of the left tenth nerve also appeared somewhat smaller.

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[1] Gaz. degli osped., No. 69, 1901.
[2] Il Policlinico, No. 44, 1901.
[3] Brit. Med. Jour., Mar. 15, 1902.
[4] Quarterly Med. Jour., Feb., 1902.
[5] Neurolog. Centralbl., No. 10, 1901.

The middle and lower part of the medulla showed a high grade of asymmetry, involving the left side. The authors believe that this case was an **example of the nervous origin of the disease,** and consider that the alterations in the left restiform body were the starting-point of the condition.

A. Pick[1] describes 41 cases of **cardiac neurosis occurring in men,**—chiefly young men, from 21 to 24 years of age,—in which he observed a moderate goiter, irritability of the heart, marked vascular irritability, some cardiac hypertrophy, inequality of the pupils, anesthesia of the pharynx, excitability of the knee-jerks, and hyperhidrosis. In some cases there was also tremor of the fingers and of the closed eyelids, and attacks of cardiac arrhythmia. Exophthalmos, and Graefe's and Stellwag's symptoms, were absent in each case. Whether the cases were **rudimentary forms of Graves's disease,** or had no direct connection with that disease, the author feels unable to state. In all the cases, however, the symptom-complex was much the same. It did not grow notably worse while under observation; and when resting, the heart-action was practically normal.

G. A. Charlton[2] discusses the **frequency and distribution of goiter on the Island of Montreal,** and particularly notes the fact that the inhabitants that drink the Ottawa water are not affected, while those that drink spring-water often show the disease. In certain regions of the city in which the inhabitants formerly drank spring-water almost entirely, but now take city water, an earlier marked morbidity with goiter has been very greatly reduced. The author believes that there is a **decided relation between the character of the soil and goiter.** If the upper layers of the soil are of hard clay, the disease does not occur; but when they are sandy and porous, it is frequent, whether the springs used are deep or shallow. The deeper layers over the whole island are composed of limestone, and thus can have but little influence upon the production of goiter.

Mariani[3] reports a rare case of **bilateral pulsating exophthalmos of spontaneous origin,** which occurred in a woman of 60. The diagnosis was spontaneous rupture of the left internal carotid into the corresponding cavernous sinus, dilation of the circular sinus of Ridley and of the right cavernous sinus, dilation of the orbital veins of both sides, bilateral pulsating exophthalmos, and left-sided blindness. The accident **occurred while straining** in defecation. The left eye was involved first. Twenty days later, without other cause, exophthalmos appeared in the right eye, and exhibited pulsation. The changes on the left side decreased somewhat. One could hear a soft systolic murmur over the whole cranium. Compression of the left carotid caused the pulsation of the left eye and the intracranial murmur to cease. Ligation of the carotid was considered, but put aside, as the condition showed great **spontaneous improvement.**

Theisen[4] reports the case of an old woman who had complained of a lump in her throat for some years, particularly noticeable upon swallowing. Examination showed a mass as large as a hen's egg, at the base of the tongue, which was soft, dark red in color, and not nodular. There was no cachexia and no enlargement of the lymph-glands. It was thought to be an **accessory thyroid gland.**

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[1] Wien. klin. Rundschau, No. 41, 1901.
[2] Montreal Med. Jour., No. 8, 1901.
[3] Il Policlinico, Sept., 1901.
[4] Albany Med. Ann., Oct., 1901.

R. Breuer[1] believes that **chronic iodin-poisoning,** as described by older writers, **is really acute thyroidism.** The symptoms were commonly observed in cases of goiter that were treated with iodin, and were practically those that Bruer and others have noted in actual acute thyroidism. He mentions, for instance, the case of a man of 56 who was given sodium iodid for goiter. The mass was considerably reduced in size; but the patient grew emaciated, became very excitable, and showed tremor and rapid pulse, the symptoms disappearing when the drug was discontinued. He also refers to a number of other cases of the same kind.

Fürth[2] reports a case of **acute iodism with a clinical picture resembling mumps.** The patient, a woman of 52, had been taking sodium iodid for 3 weeks (a teaspoonful of a solution of 1 : 30). She then showed some headache and some swelling of the eyelids. The medicine was stopped for 10 days, but was then begun again. The same day the patient had fever and marked swelling of the submaxillary glands. The symptoms decreased after the iodid was stopped, but increased again after recommencing the medication. It was evidently an iodid intoxication.

F. Reach[3] reports a study of the **respiratory gas-interchange in a subject of supposed hypothyroidism.** The patient was given thyroid in one period and the effect was compared with the previous and subsequent periods without thyroid. He determined that the respiratory interchange was decidedly increased by the thyroid, thus confirming the previous statements of Magnus-Levy and others, that **thyroid causes a decided increase of oxidative processes.**

Treatment.—Moebius[4] reports that he has used **serum from animals whose thyroids had been removed,** in the treatment of exophthalmic goiter; and that in 3 cases he noted a **marked improvement.** He suggests that the **flesh of the animal might be sufficient,** as it probably contains enough blood for this purpose. Since treatment is ordinarily without result, it would be worth while to try such measures. He looked for the possible production of albuminuria by this treatment, but found it absent.

DISEASES OF THE SUPRARENAL GLANDS.

A. Guieysse,[5] in an extensive discussion of the **histology and function of the suprarenal gland,** reports a series of investigations, and reaches the following conclusions: The suprarenal gland is a vascular gland, which is composed of two organs, the one contained within the other; *i. e.*, the cortex inclosing the medulla. In the monkey **the cortex may be divided into 4 layers,** which, from exterior to interior, are as follows: the glomerular, the spongy, the fasciculated (this being divisible into two parts, if the activity of secretion is augmented), and the reticular layer. The author was unable to find out the nature of the secretion of the glomerular layer; he considers that the **spongy layer produces a very watery liquid.** The production of this liquid is carried on in all parts of the layer, the secretion being very active toward the inner side. The fasciculated layer, particularly in its internal portion, is remarkable in that its cells contain a larger or smaller number of bodies differing from the protoplasm; the more active the secretion, the more numerous are these bodies. The secretion produced in this layer is not visible; it is probably diluted by the liquid produced in the spongy layer. The **reticular layer produces zymogenous granules; also pigment,** and the cells in it show protoplasmic filaments. The complete product of the cortex is a mixture of the secretions of the different layers. Guieysse was unable to determine whether it is modified by any secretion produced by the medulla. He could find **no evidence that the medulla has any active secretory function.** Besides the ordinary cells of the medulla, he found small collections of large nerve-cells, which represented little intraglandular ganglia. The nuclei seemed to take an active part in the production of the secretion, but no further facts determining their action could be demonstrated. He did not see direct or indirect division of the nuclei. Secretory activity is slight in the male, decidedly more marked in the female, and extremely energetic in the pregnant female; in the latter case the activity increases up to the end of gestation, then remains stationary for a time, and finally decreases. As a result of the frequent occurrence of fecundation and the consequent more or less marked increase in the activity of secretion, the gland becomes hypertrophied in the female monkey.

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[1] Wien. klin. Woch., July 19, 1901.

[2] Wien. klin. Woch., No. 45, 1901.

[3] Arch. f. exper. Path. u. Pharm., Bd. XLVII, Hefte 3 u. 4, p. 248.

[4] Münch. med. Woch., Nov. 12, 1901.

[5] Jour. de l'anat. et de la physiol., 1901, vol. XXXVII.

H. Strehl and O. Weiss[1] report the results of their work on animals, in studying the **physiology of the suprarenal bodies.** They operated successfully upon 114 animals, of various kinds [that is, the animals did not die immediately as a result of the operation]. They positively state that the **extirpation of both suprarenal glands inevitably causes death.** The animals show muscular weakness and apathy. After removing one suprarenal, the blood-pressure sinks; and after removing the second, there is a striking fall of blood-pressure. The temperature falls also. If one suprarenal is left, the animal lives, and the remaining suprarenal hypertrophies. If **one suprarenal is removed, and the vein on the other side tied,** the animal often shows the signs of double extirpation. If the vein on the other side is cut, death occurs. The latter fact indicates that the **suprarenal itself provides some substance which prevents the symptoms** that occur when the suprarenals are removed. The authors were **unable to transplant suprarenal substance.** Extract of suprarenal overcomes the symptoms of extirpation to a considerable extent if the injections are made with sufficient frequency and in large quantity; but such injections fail in their purpose, because of the production of thrombotic emboli. The latter may, perhaps, be prevented by using leech extract.

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[1] Pflüger's Archiv, Bd. LXXXVI, p. 107.

Salvioli and Pezzolini[1] carried out a study of the **different effects of extracts of the medulla and cortex** of the suprarenal gland. The extract of the **medullary portion was far more toxic** than that of the cortical; 1 cc. to 2 cc. of the first killed rabbits and dogs very rapidly, while much larger amounts of the second could be given to either animal without producing symptoms. Both extracts caused a rise in blood-pressure; medullary extract, however, produced a much more marked and prolonged rise. Cortical extract slowed the heart systole and made it stronger; medullary extract had the contrary effect. The latter also caused the respiration to become more frequent and more superficial; cortical extract had no influence upon the respiration. If the vagus was cut, or if atropin was given, cortical extract caused no change in the pulse; while medullary extract caused a marked increase in the rapidity of the already rapid heart-action.

Hamel[2] reports some observations concerning **the blood in 2 cases of Addison's disease,** one of which ended fatally and showed tuberculosis of the suprarenals. In the other case, also, there was old tuberculosis of one apex. The second case had improved greatly merely upon rest and general hygienic treatment, and had become capable of working. In the first patient, however, all treatment was useless, even the administration of suprarenal tablets. The author concludes his studies concerning the blood with the statement that **anemia is a constant and important symptom of the disease, although this is not exhibited morphologically,** the substances in the disease not being special blood-poisons. The anemia he considers to be unquestionably an oligemia. In cases of carcinoma of the suprarenals, however, a morphologic change in the blood is found; as in carcinoma of other organs, complications of the disease cause changes in the blood. If one observes anemias with morphologically intact blood in Addison's disease, this indicates tuberculosis of the suprarenals. Anemias with morphologic changes of the blood indicate carcinoma of the suprarenals or complications of the disease. Hamel found **small mononuclear cells in both cases somewhat increased,** at the expense of the polymorphonuclears.

H. Haehnel[3] reports an apparently unique case, in which there was **a combination of scurvy, Addison's disease, and scleroderma.** The diagnosis of Addison's disease was made practically entirely because of the brown spots on the mucous membrane and the marked pigmentation about the nipples and the genitalia. It is worthy of note, however, that pigmentation occurs in scleroderma.

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[1] Gaz. degli osped., 1902, No. 5.

[2] Deut. Arch. f. klin. Med., Bd. LXXI, Hefte 2 u. 3.

[3] Wien. klin. Rundschau, No. 35, 1901.

AKROMEGALY.

S. Kuh[1] discusses the **relation of the pituitary body to akromegaly,** and states that changes of some sort have been found in this body in all cases in which the diagnosis was certain, and in which the gland was carefully examined, both macroscopically and microscopically. He thinks that the **hypophysis has an influence upon the growth of the body,** and inclines to the view that diminution in the function of the gland is the cause of the disease. He describes 3 cases in which pituitary gland was administered. The first patient, a man of 29, of Jewish extraction, showed no improvement; the second, a woman of 30, showed improvement in general strength, and her headaches disappeared. After the treatment was continued a year, she had cramps in the legs, emphysema, and mental depression. The objective appearances were not altered. The third patient, a Jew of 58, showed decided improvement of symptoms, sweating and headache disappearing and his strength increasing. He died suddenly, however, in an attack of asthma, to which he was subject. The author thinks that in the last two cases **the gland had some beneficial action.**

C. L. Greene[2] reports a case of akromegaly in a patient of 25. The man had been an ice-cutter, and the author thinks that the exposure of his occupation may have been active in producing the condition. The **signs of the disease began at 20;** the hands and feet enlarged notably and the face changed in appearance. The patient also had increasing weakness. He had the general appearance common in akromegaly; but had no sensory disturbance and no disturbance of the special senses, or other disturbances referable to the general nervous system. There was no change in the clavicles, scapula, or spine. There was dulness over the sternum, which the author thought might be due to a **persistent thymus.** The most striking features were the lack of prominence of the lower jaw and the condition of the skin, which resembled myxedema. He discusses the general diagnostic features of the disease.

W. D. von Moraczewski[3] reports an investigation of the **metabolism in akromegaly** when the patient was treated with oxygen, phosphorus, and silver, and by other methods. The use of silver seemed to increase the secretion of calcium, and he believes, for this reason, that it to some extent prevented the bony growth by carrying off the material for such growth. He draws various other conclusions from this work. [There are, however, such curious errors in the tables presented that unless these are explained satisfactorily the results cannot be considered to be of value. The most striking instance of these errors is that he gives tables showing from 33 % to 67 % of amido-acids and considerable amounts of other nitrogenous substances besides urea, while at the same time there was from 87 % to 97 % of urea. The total urinary nitrogen would, therefore, amount to more than 100 % in practically all estimations that he presents, sometimes to as much as 185 % !]

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[1] Jour. Am. Med. Assoc , Feb. 1, 1902. [2] Jour. Am. Med. Assoc., Oct. 5, 1901.
[3] Zeitschr. f. klin. Med., Bd. XLIII, H. 3 u. 4

OSTEOMALACIA.

Schmidt[1] has used the **diaphoretic treatment of osteomalacia** for the purpose of causing an intense diffuse hyperemia of the skin, and, in that way, relieving the overfilled blood-vessels of the periosteum and bone-marrow. Possibly it also increases the excretion of organic acids in the sweat, and produces good results in this way. He describes the details of his method of giving sweat-baths.

I. His[2] reports a study of the effect of phosphorus in osteomalacia, based upon an investigation of the calcium excretion. He finds that **phosphorus causes any retention of calcium** to increase to about 15 times the previous amount; and that, therefore, phosphorus must be considered to have a definitely favorable influence upon osteomalacia.

DISEASES OF THE BLOOD.

CONDITIONS AFFECTING THE CONSTITUTION OF THE BLOOD.

A. Pappenheim[3] discusses the results which he has obtained in examining the **blood of hibernating animals,** before hibernation, during hibernation, and directly after their awakening. The animals which he used for this purpose were 8 marmots, 3 of them less than a year old, the others older. His purpose in making the examination was to see whether he could not learn something of the **changes which normally took place during blood regeneration,** as animals during hibernation would evidently need less blood and less tissue in general during hibernation, would therefore reduce their blood-supply during this period, and would require a greater amount of blood on awakening. He first discusses the methods of staining, and states that the best results were obtained by staining with hematoxylin and counterstaining with tri-glycerin mixture. Good results were also attained with methylene-blue or toluidin-blue and counterstaining with methylene-green triacid mixture. He first describes the condition of the blood of the marmot under normal conditions, and then states that during hibernation he found that the animals became much emaciated; lost much weight; acetone appeared in the urine; the **spleen became very greatly increased in size,** and there was a **marked deposit of hemosiderin and many cells containing red blood-corpuscles.** The long bones showed **fatty change of the marrow** in the parts that were previously red and in the marrow of the ribs there was marked deposit of hemosiderin. The blood showed **no nucleated cells; no noteworthy leukocytosis, reduction of hemoglobin, or hydremia,** and there was no evident thickening of the blood. Except for the macroscopic changes in the marrow there was no marked alteration in the blood itself. He considers that the **anemia is a secondary one, and is due to the sleep.**

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[1] Wien. klin. Woch., No. 27, 1901.
[2] Deut. Arch. f. klin. Med., Bd. LXXIII.
[3] Zeitschr. f. klin. Med., Bd. XLIII, Hefte 5 u. 6.

The anemia goes hand in hand with a reduction of the body-fat and body-albumin, and the same is true of the changes in the bone-marrow. In the regeneration of the blood the same changes were seen whether the animals were awakened naturally, or by placing them in a heated chamber. They were examined a few hours after awakening. There were no nucleated red cells in the blood; as a rule, the blood seemed somewhat hydremic; the marrow became typical red marrow, the reddening appearing centrifugally, while the change to red in the marrow when hibernation came on had taken place in exactly the opposite direction. In an old marmot the marrow showed a majority of red cells, and the white cells were chiefly mononuclear forms. There were numerous nuclear divisions seen. There were also large numbers of normoblasts. The latter were much fewer in the young animal. The condition in the young marmot was about the same as before sleep, but in the old marmot there was marked evidence of regeneration in the bone-marrow. In other words, the **blood of the old animal was insufficient for its function after the period of hibernation.** Pappenheim then gives a **classification of anemias in general,** dividing them into two main classes: primary hemophthisical, and primary myelophthisical. Under the first heading he makes the subdivisions: (*a*) those in which the blood-destruction is normal in character but increased in amount; (*b*) those in which there is an abnormal method of blood-production. The first are traumatic anemias; the second toxogenic. The latter includes pernicious anemia, pyrodin and arsenic poisoning, and other anemias of intoxication; gastric carcinoma, syphilis, etc., and the anemias due to the bothriocephalus, the anchylostoma, and to pregnancy. Under the second main heading he makes several subdivisions: (*a*) Simple traumatic extirpation of the marrow—a very rare condition except when several limbs are removed coincidently. (*b*) More or less general substitution of the marrow by lymphatic overgrowth, as in lymphatic anemias, myelogenous pseudoleukemia, etc. (*c*) Heterogeneous multiple circumscribed neoplasms, which substitute part of the bone-marrow and cause intoxication of the remainder, as in carcinoma. (*d*) Direct specific intoxications of the bone-marrow itself, of natural or experimental character, the effect of which upon the blood has not yet been accurately determined. In the first main class the essential difficulty is increase in the blood-destruction. In the second main class the essential difficulty is reduction in the size or capacity of the blood-building organs.

Brandenburg[1] discusses the **alkalinity and the alkali tension of the blood in disease.** The meaning of alkali tension is as follows: If blood be placed in a dialyzer, and an alkaline fluid in the outer vessel, the latter fluid can be made of such strength that neither this fluid nor the blood will alter its original alkalinity. The alkalinity of this fluid indicates the alkali tension of the blood. The determination of the mere alkalinity of the blood is of very doubtful value. In most cases it varies directly with the concentration of the blood. In order that it may be of any value, the author believes that there **should be a coin-**

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[1] Deut. med. Woch., Jan. 30, 1902.

cident determination of the total nitrogen. This will indicate the albumin-content of the blood, and will show whether the alkalinity is diminished merely because there is a diminution in the concentration, or whether there has been an actual increase of acid. The diffusible alkali of the blood stands in a proportion to the total alkali of about 1 : 5. In general, the proportion increases with the decrease in the albumin concentration of the blood, and decreases with increase in the albumin; therefore, the amount of diffusible alkali is largely dependent upon the amount of albumin in the blood. It is a notable fact that when small quantities of alkali are added to the blood, the proportion of diffusible to total alkali does not change. The diffusible alkali varies inversely as the total alkalinity of the blood. The normal alkali tension equals about 60 mg. of NaOH, the standard total alkalinity (determined by the Loewi-Zuntz method) being considered to be 300 mg. of NaOH. Increase of carbonic acid in the blood causes an increase in the diffusible alkali, since the CO_2 combines with the alkali that has previously been combined with albumin, and, hence, brings this alkali in solution as alkaline carbonate; consequently, the diffusible alkali is greater in amount in the veins than in the arteries. In typhoid fever the alkali tension was found above normal; in pneumonia, somewhat decreased; in uremic coma, decidedly decreased. The freezing-point varied, with considerable regularity, directly as the amount of diffusible alkali; consequently, the diffusible alkali is probably the chief factor in depressing the freezing-point; and, hence, depression of the freezing-point does not necessarily always indicate retention. As to the **importance of the alkali tension,** it is probable that it has something to do with the susceptibility to infection, since the diffusible alkalies have a bactericidal action; also, in acid intoxications the alkali tension will probably be found reduced.

T. Houston[1] contributes a study of **the blood in various forms of edema,** particularly in that of anemia. He believes that he has demonstrated that the absence of loss of weight and the apparent absence of emaciation in anemia are due to an abnormal accumulation of fluid, and that the first stage in the cure of such anemic conditions is getting rid of this excess of fluid. In pernicious anemia a gain in weight without an improvement in hemoglobin is an unfavorable sign, indicating an increased deposition of water in the tissues. Sometimes, however, this is immediately followed by a marked improvement; otherwise, the patient usually dies soon. The **edema of anemia** the author considers to result from hydremic plethora of the blood, and to be different from the edema found in Bright's disease. He thinks that a study of the body-weight and of the hemoglobin in other conditions associated with edema may give important suggestions concerning the origin of the edema.

Türk[2] exhibited before the Vienna Medical Society a patient with **splenomegaly, cyanosis, and an enormous increase in the blood-elements,** the erythrocyte-count being 9,150,000; the leukocytes, 23,000 to 26,000; and the specific gravity of the blood, 1.070. It was a case, apparently, of true **overfunctionation of the myeloid system,** particularly of the erythroblastic apparatus, with coincident evidence of irritation of the leukoplastic apparatus.

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[1] Brit. Med. Jour., June 14, 1902.

[2] Centralbl. f. innere Med., No. 12, 1902, p. 305.

Saundby and J. W. Russell[1] report a remarkable case of **unexplained chronic cyanosis** in a man of 48, and discuss the 3 other cases that they have been able to find in the literature. The main points that the cases present are dyspnea, headache, constipation, a tendency to hemorrhage, some enlargement of the liver, marked enlargement of the spleen (in 3 cases), a trace of albumin in the urine with a few casts, some pigmentation (in 2 cases), and in all the cases a **remarkable increase in the red blood-corpuscles and hemoglobin,**—particularly the former,—with an intense cyanosis. All the patients were over 45 years of age. Postmortem examination in the case reported showed no cardiac lesion. The liver, spleen, and kidneys were enlarged and congested, the spleen weighing 48 ounces. The suprarenal capsules were small, soft, and dark red. The bone-marrow looked normal. There is no microscopic report of the organs. The enlargement of the liver and spleen in all the cases seemed to follow the onset of the cyanosis.

P. Reckzeh[2] contributes an extensive report of his **studies of 10 cases of measles and 10 of scarlet fever as to the condition of the blood,** giving a very complete bibliography as well. He determined the number of the red and the white cells, made morphologic studies of these cells, determined the hemoglobin, roughly estimated the time of coagulation, etc. The main points concern the conditions relating to the leukocytes. The author especially insists upon the importance of hypoleukocytosis in measles and of hyperleukocytosis in scarlet fever. He finds that the latter, in most cases, occurs almost at once, and usually—contrary to Türk's statements—is likely to reach its maximum very rapidly. In doubtful cases **these contrary conditions in the leukocytes are very important in diagnosis.** In both diseases complications cause a rise in the leukocytes. Nephritis, however, which occurred in 2 cases of scarlet fever, apparently had no influence upon the leukocytes. Reckzeh also **directs especial attention to the eosinophiles in the two diseases.** In scarlet fever these cells show an almost immediate rise, and persist at a high point throughout the whole eruptive period; while in measles they are very low, and usually do not rise until about 2 weeks after the onset of the disease.

M. Oppenheim and G. Löwenbach[3] report some investigations of **the blood in constitutional syphilis,** under the influence of mercurial treatment, with especial reference to the amount of iron. In the patient examined, who was in the early stages of the disease, they found the hemoglobin reduced to between 65 % and 85 %. Jolles's ferrometer showed a corresponding condition of the iron. The number of red cells was about normal, and the whites showed only physiologic variations. No iron was present in the blood-serum. The use of mercury by injection or inunction had no notable influence upon these conditions.

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[1] Lancet, Feb. 22, 1902.

[2] Zeitschr. f. klin. Med., Bd. XLV, Hefte 1–4.

[3] Deut. Arch. f. klin. Med., Bd. LXXI, Hefte 4 u. 5.

G. Strube[1] insists that in carrying out methods for the **determination of the nature of blood and albumin by biologic procedures,** it is, in the first place, exceedingly important to **determine the action of the precipitating serum upon both homologous and heterologous serums.** He says that presumably **specific serums do actually precipitate heterologous serums,** even in dilutions as great as about 1 : 100. He says, for instance that a serum, presumably specific to human serum, produced precipitation in the serum of monkeys, guinea-pigs, hens, pigs, calves, dogs, goats, and sheep. It acted, however, on human blood in very great dilutions; and on the other blood, only in dilutions of about 1:50, or at most, 1:100. The author states that by injecting intravenously, one can produce an active antiserum very rapidly; and that the activity of the serum is usually greater when produced in this way. He injects 6 times a week, using 1 cc. He finds that about a week after the last injection the maximal activity of the serum is reached. After about 4 weeks it begins to decrease in activity, and ultimately the activity disappears entirely. These animals can then be again injected, and usually provide a more active serum than before. Strube states that he **has preserved a serum,** either with or without carbolic acid, for as long as three months, and found its action unchanged. The best conditions are obtained by using a 0.5 % blood solution, the blood being diluted with 0.8 % sodium chlorid. A control with normal serum of the same kind should always be carried out, and the test should be observed for several hours. When **working in this way, the author has never seen a reaction with any but specific serums** in a dilution greater than 1:100. He compares the conditions concerning the precipitin reaction with those concerning the agglutinin reaction in the diagnosis of typhoid fever, and thinks that much the same precautions are necessary in drawing conclusions in either case. He has investigated the effect of the injection of certain forms of albumin upon other varieties of albumin, and has found that the result upon the serum is apparently entirely specific; but the results previously mentioned show that it is not absolutely specific. The fact **that specific antiserums will act to some extent upon heterologous serums is probably to be explained upon one of two bases:** Either the albumins in these serums are distinct, but very closely related; and the serum that is specific to one form acts in weaker degree upon those most closely related, like the agglutinating bodies acting upon the typhoid and colon bacilli; or, on the other hand, there is a series of substances in the injected serum and in the serum of the animal into which it is injected, and these are identical in animals of the same species, throughout the whole series of substances; while in animals of other species, some of these substances, but not all, may be the same. Therefore, an imperfect reaction may be due to a reaction between certain members of these series of substances. Strube believes that the **precipitation is not due to the action of an enzyme,** because he has convinced himself that antiserum has a quantitative action. He has attempted to precipitate out specific forms of albumin by using **fractional precipitation with various kinds of antiserums;** he has, however, been unsuccessful.

[1] Deut. med. Woch., June 12, 1902.

E. Zeimke[1] contributes a further article on the specific reaction of blood-serum, and states that since there is so much difficulty in obtaining human blood-serum to be used in treating the rabbits, he used **blood taken from dead bodies,** and found that even when the blood is 3 or 4 days old, it still gives excellent results. He has had no instances of infection of the animal experimented upon through using such serum, even when the person from whose body the blood was taken had been dead for more than 4 days. He recommends, however, that the serum be injected subcutaneously rather than into the peritoneal cavity, as infection would be more likely to be avoided in this way. The injections were carried out every 3 or 4 days, and, in all, 60 cc. to 100 cc. of serum was used. In obtaining the serum from the animal he used the following method. The animal is tied upon its back, the carotid on one side is laid free, isolated, tied in 2 places, and then, either by introducing a glass cannula or by cutting directly across the carotid, the blood is allowed to flow directly into a sterile vessel. After it is defibrinated it is placed in sterile test-tubes and kept on ice, after which it is pipetted off. One important point is, if possible, to provide some method of keeping the serum, or the active part of the serum. It is known that the serum of the animal loses its activity to a certain extent if preserved. Other methods than using chloroform have been suggested, but have not been wholly satisfactory, and Zeimke tried the plan of **saturating the serum with ammonium sulfate,** collecting the precipitate which contains the globulin, drying this over sulfuric acid, and then, when needed, dissolving in physiologic salt solution. This still gave the reaction after 3 months, but was not so active as fresh serum, and he considers that we have at present **no method of preserving the agglutinins in satisfactory condition.** One point which he adds to previous technic is that the use of **0.1 % soda solution is better than physiologic salt solution** for dissolving the dry blood to be tested, as soda extracts better than salt solution. This solution should not be stronger than at most 1 %, however. He thought it possible that human blood that had been smeared over with animal blood might not give a clear reaction, but he tried mixtures of human and animal blood and found that they reacted typically with the serum of an animal treated with human blood-serum. He found the **employment of potassium cyanid useful** as a method of extracting old stains, if the extract were subsequently neutralized with tartaric acid in substance. The tartaric acid must be added or the cyanid will interfere with the reaction.

S. Minovici[2] states that after he has made a certain number of injections, he tests a small amount of blood from an ear-vein. If it is active, he chloroforms the animal, opens the thorax, brings the still-beating heart into a vessel, opens it there, and collects all the blood in the vessel. In this way he gets about 50 cc. from one rabbit.

Uhlenhuth[3] has carried his investigations concerning the specific serum reaction in another direction, and now reports that by this means he is **able to differentiate between different kinds of meats;** and he believes that this will become an important method of carrying on meat inspection. Animals injected with the extract of meats from other animals, subsequently have substances in their blood-serum that react only with the meat extracts from the flesh of the same species of animals as that previously used in the injections.

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[1] Deut. med. Woch., Oct. 17, 1901. [2] Deut. med. Woch., June 12, 1902.
[3] Deut. med. Woch., Nov. 7. 1901.

Conradi,[1] considering autolysis of organs to be closely analogous to the processes which go on in the breaking-down of protoplasm, and having in mind the fact that the liver seems to provide the anticoagulant substances after the use of peptones, etc., investigated the juice of livers that had undergone autolysis, and of other organs, both *in vitro* and *in vivo.* He then investigated the juice obtained by pressure (Buchner's method) in the same animals. He decides that the **pressure juice of organs contains substances which actively hasten coagulation.** The **juice from the autolyzed organs contains anticoagulant substances.** These substances he considers different in their chemical and physical properties, and especially in the fact that the coagulating substance is more closely united with the parenchyma of the cells than is the anticoagulant; hence, **coagulation of the blood must be explained through the destruction of the morphologic elements** which occurs with every loss of blood,—this including the erythrocytes. As a result of this cell destruction, the coagulant substances are set free. The anticoagulant is not collected in the blood and is not produced by the blood, but is developed by the parenchyma of the organs. The author does not find that the two substances act in a directly antagonistic manner to each other. Probably they have different methods of action, and perhaps they may both act at the same time.

H. Strauss and W. Wolff[2] discuss the **hemolytic action of serous fluids,** and find that **transudates have a much less marked hemolytic action** than the blood of the same individuals, while exudates approach the blood-serum in their activity. The amount of the various salts in the effusions has little influence, but the hemolytic activity seems to be in direct relation to the nitrogen-content. The active substance is not dialyzable, and the reduction of activity in the transudates is due to the absence of the substance destroyed by heating to 55° C. (133° F.).

J. Mannaberg[3] states that he has determined that the **serum of animals drowned in water is stained more or less red, as the result of hemolysis.** If the animals are drowned in physiologic salt solution, hemolysis does not occur. It may be produced by the injection of large amounts of water into the stomach and intestine. The author repeatedly found a more marked hemolysis in the portal vein than in the blood of the right heart; hence, he decides that the liver retains the dissolved hemoglobin. This observation is of interest in connection with the relation of hemoglobinemia to icterus.

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[1] Hofmeister's Beiträge zur chem. Physiol. u. Pathol., 1901, Hefte 3 u. 4.

[2] Fortschritte der Med., Nos. 1 and 7, 1902.

[3] Wien. klin. Rundschau, No. 41, 1901.

The Red Corpuscles.—E. Hoke[1] reports some investigations of the **nitrogen-contents of the red blood-cells** in severe secondary anemias, in chlorosis, and in pernicious anemia. He finds that in chlorosis and secondary anemias the amount of proteids decreases; in pernicious anemia the contrary is true: *i. e.*, the normal nitrogen content of the red cells is about 5.52 %. The cases of pernicious anemia showed 6.59 %; a severe secondary anemia and endocarditis showed 5.5 %; 3 cases of chlorosis showed from 3.3 % to 3.9 %.

W. A. Campbell and H. W. Hoagland[2] contribute a study of the **blood-count at high altitudes.** They find that the blood-count is increased 250,000 to 500,000 per cmm. in Colorado Springs, as compared with sea-level; and to about the same degree by ascending from Colorado Springs to the summit of Pike's Peak, by railroad, without exertion. They also find that active exercise (running) decidedly increases the blood-count; that massaging and rubbing one arm cause an increase in the count on that side, as compared with the other; and, likewise, that other causes of vasomotor change have the same effect, particularly heat and cold locally applied. A series of 10 cases were counted before and after ascending from an elevation of 6000 feet to one of 14,147 feet, and after descending to the original level. The blood-count rose while at the higher level, and again decreased to the former point, or below, after the descent to the original level. The authors also studied rabbits, counting the **peripheral blood and blood from the mesentery.** They found an increase in the peripheral blood, but no increase in the blood from the mesentery. They believe, therefore, that the increase in the count is **due to changes in the distribution of the blood.**

L. Mohr[3] discusses **6 cases of poisoning with benzol chlorid** which he has recently observed. In all, it was an occupation intoxication. The notable facts about these cases are that in all the instances but one the **poisoning appeared after the excessive use of alcohol.** It is known that alcohol is dangerous for workers in anilin dye factories, as it apparently dissolves the nitrobodies and makes them more easily absorbed. The most striking points about these cases were the changes in the blood. Degenerative changes were found—*i. e.*, fragmentation, very marked change in the size and shape, and marked alterations in the staining of the red blood-corpuscles. One striking change was that the stroma often stained badly, while in the interior of the cell there was a nucleus-like body—the condition which Ehrlich has called **hemoglobinemic degeneration.** Regenerative changes were seen in the presence of nucleated red cells. **In all the cases met hemoglobin was found in the blood.** Brat has reported the presence of **glycuronic acid in the urine** of such persons, considering its presence to be a warning to remove the patient from the danger of further poisoning. This substance was looked for in 2 of Mohr's cases, but was absent in one; and there was, at most, but little in the other. He also looked for **alimentary glycosuria** in 2 cases, and found it absent. **Hematoporphyrin was present in all.** Some noteworthy symptoms were the presence of marked muscular tremor in 2 cases, optic neuritis in another, and profound soporosity in all. The author believes that oxygen inhalations did good, but considers that this was not due to a direct increase in the oxidative processes, but to the fact that it made the patients breathe more regularly and more fully, thus producing better oxygenation of the blood.

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[1] Zeitschr. f. Heilkunde, 1901, Hefte 8 u. 9.

[2] Am. Jour. Med. Sci., Nov., 1901.

[3] Deut. med. Woch., Jan. 30, 1902.

E. Grawitz[1] **divides hematolysis into two different processes**: In one the red corpuscles in the circulation are left intact, but poisons exert an influence upon the liver, spleen, and bone-marrow, which causes the red cells to be destroyed in excessive amounts in those organs; in the second form of action the red cells are actually destroyed in the circulation, and produce hemoglobinemia, etc. The **first action is called plasmotropic; the second, plasmolytic.** One of the most distinct examples of purely plasmotropic action is lead-poisoning. Lead produces marked basic degeneration of the red cells; but, even in the most severe cases of lead-poisoning, hemoglobinemia and other signs of destruction of the red cells in the circulation are unknown. Further investigations have indicated to Grawitz that **when hemorrhages occur in the gastro-intestinal tract, basic degeneration of the red cells occurs with great frequency,** and that this shows direct dependence upon the hemorrhages. He believes that this indicates that products of the blood, when absorbed, act as poisons. In order to prove this, he first administered blood through the digestive tract to animals; but, in the quantities in which it could be used, he found no definite result. Larger amounts he did not use, because he feared the influence due to the effect of one species of blood upon another. When, however, he administered large quantities of hemoglobin preparations to human beings, he found that decided basic granulation of the red cells appeared; and that the granulations increased in number with increase of the dose, and disappeared soon after the use of hemoglobin was stopped. He believes that this positively indicates that the **passage of products of the breakdown of blood into the circulation causes a plasmotropic action** upon the red blood-cells, and that the products of blood destruction are, therefore, toxic. This, he thinks, is a satisfactory explanation of the anchylostoma anemia. He considers it probable that the parasites themselves do not produce a poison, but that the constant losses of blood which they cause constitute one factor in the production of the anemia, and that another factor is the absorption of blood-products which act as plasmotropic poisons.

C .Y. White and W. Pepper[2] report some clinical and experimental investigations concerning the **basophilic granulation of the erythrocytes.** They examined cases of chronic lead-poisoning, workmen in lead-works who had none of the ordinary symptoms of lead-poisoning, persons who had been subjected to excessive local or general applications of heat, and animals experimentally subjected to lead-intoxication. Four cases of typical lead-poisoning showed the granules regularly; both coarse and fine granules were present, sometimes together in the same cell. The number of granulated cells was in fairly direct proportion to the severity of the disease, and the granules decreased with improvement in the symptoms. In severe cases coarse granules were more common. Except for the granulations, the blood was nearly normal; although the cells were somewhat irregular and pale, and a few nucleated cells were found. The nuclei of these were intact. Twenty-one persons who had worked in lead, but who had no symptoms of poisoning, showed granules in all cases. In 2 instances the men had been working with lead for only 4 days; in 1 case, for as long as 24 years. With one exception, these persons had never had symptoms of lead-poisoning. Of these persons, 12 had slight poikilocytosis; 2 had reduction in hemoglobin; 3, polychromatophilia; 5 showed normoblasts. Men who had **worked in excessive temperatures,** persons who **had had heat applied locally** for therapeutic purposes (300° F.), and those who **had had lead-water and laudanum applied** to the unbroken skin, showed no granules. **Dogs were given gram doses of lead acetate,** and showed granules, usually in about 3 days; and indistinct granulations were noted after 24 hours. The granules in dogs' blood tended to clump; this did not occur in that of the lead-workers. One of the authors took **7.5 grains of lead acetate, and found granules in his blood** about 24 hours afterward. The blood from the portal and splenic veins in one dog showed marked granules in the blood from the mesenteric vessels, the heart, and an ear vein. In 5 other instances this difference was less marked. Granules were found in spreads from the bone-marrow, but they may have been in the circulating blood; and the granules were never found to be excessive in number. Sections from the bone-marrow showed no granules. The methods of staining which the authors recommend are hematoxylin and eosin, and, particularly, theonin phenique. They fixed with heat for a few moments. Bone-marrow spreads were fixed by other methods, because they contained so much fat. The authors decide that the granules are constant in lead-poisoning, and appear even before subjective or objective symptoms can be found. **Working in lead does not produce an immunity,** as demonstrated by 2 persons, one of whom had worked for 24 and the other for 20 years in lead, and had no lead-poisoning, and yet showed granules. They consider the granules to be true degeneration products.

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[1] Deut. med. Woch., Dec. 26, 1901.

[2] Am. Jour. Med. Sci., 1901.

Jawein[1] discusses the **basophilic granules in relation to a case of bothriocephalus anemia** of the progressive pernicious type. The blood at first showed 1,290,000 red cells, 25 % of hemoglobin, and a specific gravity of 1.036. There were numerous megaloblasts, a few microblasts, and slight poikilocytosis. Drugs administered at once, brought the worm away. The patient, however, developed pneumonia. The blood condition grew much worse; a crisis occurred; and with the crisis, the blood-count rose from 577,500 red cells and 11 % of hemoglobin, to 1,444,000 red cells and 26 % of hemoglobin, in 3 days. Megaloblasts diminished. The nuclei of the new cells looked very irregular; several pieces of the nuclei were often found lying isolated from the nucleus. The latter often looked like a network. Frequently a number of granules were seen, arranged rather irregularly, giving an appearance as if the nucleus had been blown into dust. The granules were sometimes strewn irregularly throughout the cell. There were all stages, from the finest to the coarsest granules. In the further course of the convalescence the changes in the blood became less marked. There was improvement in the appearance of the blood for about 10 days after the crisis; afterward the nucleated cells and the megaloblasts gradually disappeared, and the blood became about normal in appearance. The author decides that **basophilic granulations and polychromatophilic degeneration occur only in young red cells, and indicate regeneration.** The basophilic granules arise from karyorrhexis. Polychromatophilia is due to solution of a portion of the normal substance in the protoplasm of the red cell. The appearance of either basophilic granules or polychromatophilia indicates increased function of the bone-marrow, and shows that the administration of arsenic and such drugs is not indicated. The author disagrees with Grawitz that blood-poisons can cause granulation of the cells, and thinks that the observation of an intact nucleus together with granules does not speak against the origin of these granules through karyolysis, as he has seen a red cell with 2 nuclei—one normal and the other undergoing degeneration.

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[1] Berl. klin. Woch., No. 35, 1901.

E. Bloch[1] contributes a long article concerning basophilic granulations of the red blood-cells, and other changes resembling them, and also discusses their nature and the probable ways in which they are produced. **He has produced granulation in red blood-cells by intravenous injections of methylene-blue into mice.** He believes that the granulations produced by this method, as well as those produced by similar methods, are due to the action of the stain upon the red blood-corpuscles, and the **precipitation of substances contained in the red blood-corpuscles.** In other words, it is not a vital staining, and the **granulations are not pre-formed bodies.** He has investigated the effect of potassium chlorate, concentrated salt solution, pyrogallol, and pyridin on the blood-corpuscles of warm-blooded animals; with the exception of the latter substance, these poisons all produced the same effect in acute or chronic poisoning. The different results obtained by different observers, or by the same observer in different experiments, he attributes to the amount and concentration of the poison, or to the varying resistance of the different animals. After using pyrodin or phenylhydrazin, or other derivatives of this latter substance, he believes that there is a change in the protoplasm, which at first is evident only by the use of stains, and which is due to precipitation of some of the substances contained in the cell which are, under normal circumstances, in solution. If the poisoning is more marked, however, hemoglobinemic bodies appear within the cells. The granulations which are seen at first only when stains are used, are the **preliminary effects of the hemoglobin degeneration.** These changes are not merely mechanical results, for they do not occur after even direct addition of concentrated solution of the blood. As to the diagnostic and prognostic importance of basic granulations of the red blood-cells, he opens his discussion by making the statement that there is **scarcely any diagnostic or prognostic importance to be attached to them.** They have a theoretic importance and some clinical interest, but their importance in the latter connection is not very great. They are evidence of beginning changes in the blood, and if other changes are not shown by other methods, they are of importance in indicating that decided change has occurred, and if, for instance, a patient is seen with marked abdominal colic and without any other distinctive signs, the discovery of considerable numbers of basic granules would speak in favor of the existence of lead colic. He believes, however, that a negative result of an examination is of no importance, for he has frequently found them absent in lead colic. He has usually found them present in pernicious anemia, but they may be absent. The same is true of carcinoma. So far as his experience has yet gone, they are not present in sarcoma, probably because decided anemia is not common in sarcoma. He has examined a considerable number of posthemorrhagic anemias, and was struck by the fact that the granulations were very rare. Suppurative processes are sometimes accompanied by a considerable number of basic granulations, but in such conditions they are often absent. He believes that these **granulations are not due to the fever itself,** as in most conditions accompanied by fever they are not present. In leukemia he has found them variable—sometimes present, sometimes absent. As yet, he has never seen them in chlorosis; in 3 cases of hemorrhagic diathesis of unknown cause they were absent. He has **found them in a large number of young children,** chiefly in splenic enlargement with anemia, and usually in cases which showed decided signs of rachitis. In congenital syphilis he has not found them. In cirrhosis of the liver, in dysentery, in 2 cases of acute yellow atrophy of the liver, and in 2 cases of pseudoleukemia they were absent. He found them **in about 50 % of his cases of lead-poisoning,** chiefly in the more acute or recent cases. They are frequently absent in old lead-poisonings, and he does not believe that their absence is of any diagnostic significance, while their presence is of the significance previously spoken of. He does not believe that the granulations are due to the gastrointestinal disturbances, however. They may be entirely absent in patients who have marked gastrointestinal disturbance even when they have lead-poisoning, and they are, as a rule, absent in other kinds of gastrointestinal disturbances. They also do not disappear after the disappearance of the gastrointestinal symptoms in lead-poisoning. It is, however, true that they are most commonly found in cases of lead-poisoning with colic. He has given rabbits lead, and has only rarely found the granulations in their blood-corpuscles. Other authors have had different results, probably as a result of their using animals of different susceptibility. **In other poisonings in human beings he has had negative results,** though he has examined a considerable number of cases of poisoning with mercury and potassium permanganate. Strauss, however, has reported their presence in acute atropin-poisoning. He has examined 4 cases of malaria, and found them absent. Discussing the origin of these granulations, he states that he believes they are **due to degeneration of the protoplasm,** and are not derived from nuclear destruction. Against their originating from the nucleus are 4 main points: the fact that one may often observe nuclear divisions and large numbers of granulations in the same blood-corpuscles; the general staining properties of these granulations; the absence of granulated red cells, or of their preliminary stages, in the blood-building organs; and, further, the whole character of karyorrhektic processes, and the manner of disappearance of the nucleus, in general, speak against their origin from the nucleus. He gives an extended discussion of the manner in which the nucleus disappears from the red blood-corpuscles, and concludes that, as a rule, it is cast out of the corpuscle. As to the **nature of the granulations** he expresses no definite opinion. He has found the following facts, however: Fixation by the heat is not necessary to demonstrate them, for they appear after merely drying in the air and staining with a concentrated dye. They are not changed by being kept in absolute alcohol for days; they are not soluble in ether, but are in potassium hydrate; they become obscure after exposure to weak acetic acid. They are apparently not acted upon by distilled water.

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[1] Zeitschr. f. klin. Med., Bd. XLIII, Hefte 5 u. 6.

J. Sabrazes and Bourret[1] report a case of **attempted suicide by taking lead-acetate solution,** which contained a small amount of arsenic. Most of the poison was vomited immediately after taking. The patient was at once brought under treatment, and the amount of lead absorbed was undoubtedly very small. Nevertheless, colic, the blue line, headache, and marked blood-changes occurred, the latter consisting in the appearance of **normoblasts, basophilic granules, and polychromatism.** There was also a neutrophile polynuclear leukocytosis, which appeared on the second day. This increased with the other toxic symptoms, up to the fifth or sixth day; and then there was a gradual decrease, as the other symptoms also decreased. An intercurrent attack of measles caused a decided increase of the polynuclear neutrophiles. The small amount of arsenic taken was considered to have had no influence in the production of the basophilic granulation. In a large series of cases in which **arsenic or cacodylic acid** was used in large quantities as medication, no such changes could be observed. The authors insist upon the importance of basophilic granulation as an evidence of lead-poisoning.

O. Montz[2] examined 32 persons engaged in occupations that exposed them to **lead-poisoning.** Of these persons, 26 had no trace of actual lead-poisoning; 5 had had neuralgia and other symptoms; and 1 had distinct lead paralysis. All but 5 of the total number showed basophilic granulation. The extent of the degeneration bore some relation to the length of time that the workers had been employed.

W. Löwenthal[3] has investigated the **effect of tin and cerium in the production of basophilic granulations** of the red blood-cells. He found that after the use of chlorid of tin and sulfate of cerium, considerable numbers of granules did appear in the blood. Several of the animals, however, had peritoneal infection. He noticed such a striking difference in the results in different animals that he thought that the individual animals might show different tendencies to the granulations, and investigated the question whether the hygienic arrangements for the animals had anything to do with the granulations. He found that animals that had been kept in the open air, protected from wet and cold, and well fed, rarely showed the granulations without some experimental procedure; while of 8 other animals that were apparently normal but had just been bought in the market, 7 showed the granulations at the time of their purchase. The author, therefore, considers that any **results obtained in such sensitive animals as guinea-pigs are subject to great question,** and he is in doubt as to whether the cerium and tin caused the granulations which he found. Guinea-pigs tend to have these granules in their blood, even under fairly normal circumstances; and they certainly tend to appear if the hygienic arrangements are not satisfactory.

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[1] Gaz hebdom. des sci. méd. de Bordeaux, No. 39, 1901.

[2] Petersb. med. Woch., July 13, 1901.

[3] Deut. med. Woch., April 10, 1902.

Guyot[1] reports the presence of **basic granulations in cases of hemoglobinuria due to cold.** The number was in direct relation to the intensity of the attack.

The Leukocytes.—Carstanjen[2] discusses the **relative percentages of the various forms of white blood-corpuscles in human beings under different normal circumstances;** more particularly, at different periods of life. He found that **directly after birth** and during the first 24 hours of life the polynuclear cells reach nearly 74 %, and that from that time until about the ninth day they decrease rapidly, and reach about 36 %, maintaining this point throughout the **first half-year of life,** and then gradually **increasing up to the fifth year.** From this period up to old age, they show moderate, irregular variations, between 50 % and 70 %. The **lymphocytes show low values on the first day of life,** and increase decidedly up to about the twelfth day. After the first six months, up to the fifth year, they gradually become reduced in numbers, and subsequently show irregular variations. The **transitional forms are very high** during the first week of life, they then decrease until the end of the sixth month; and from this period on, they vary from 6 % to 9 % or 10 %. The large mononuclear leukocytes and the eosinophiles do not vary greatly at different periods of life. As to the **changes at different times of day,** the author decides that the number of polynuclear leukocytes is generally higher before a meal than a few hours after it, although a short time after taking food they are likely to increase. They reach their minimum, however, 3 or 4 hours after taking food, and then increase again. The lymphocytes follow directly the opposite course. Transitional forms and eosinophiles show no changes related to food. The percentage numbers of polynuclears and lymphocytes show marked changes during menstruation, but there are no characteristic conditions. The author found, in comparing pregnancy, the very beginning of the puerperium, and the second week of the puerperium, that the conditions before labor and a week later were about the same; but that directly after labor the polynuclear cells are much increased, and the lymphocytes, transitionals, and eosinophiles decidedly reduced. The percentage value of the different forms of leukocytes, under ordinary normal circumstances, does not greatly vary. Carstanjen also finds that the **blood taken from different portions of the body** shows about the same relation between the cells. In the early days of life, however, the variations are decidedly greater; and less marked, but still striking, variations are found during all the early years of life.

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[1] Gaz. degli osped., No. 27, 1902.

[2] Jahrb. f. Kinderheilk., Bd. LII, Hefte 2 to 4.

W. Hannes[1] refers to Bohland's previous work, which showed marked **changes in the number of leukocytes after using hydriotics and antihydriotics.** Hannes had similar results from **simple sweating without the use of drugs.** He examined 29 children during the course of free sweating, and in the interval, the sweating in some instances being spontaneous, in others the result of hot-air baths and similar procedures. Of the children, 77% showed **decided leukocytosis during the sweating,** while 5 showed normal conditions or leukopenia. He was careful to exclude children who showed any infectious disease or other condition which of itself was likely to cause an increase in the leukocytes; indeed, some of the children were perfectly healthy. In those **cases in which leukopenia occurred** he made the interesting observation that a **high grade of rachitis was always present.** The effect of food and of other physiologic factors which influence the leukocyte count was so far as possible excluded. The increase in the number of leukocytes was usually from 3000 to 5000, and leukocytosis was not considered to be present unless the number rose above 13,000. Hannes believes that this leukocytosis was due to a positive chemotactic action of certain substances which are always present even in the normal body, but which occasionally act upon the leukocytes when certain factors, such as increase in temperature and the like, make it possible for them to do so.

A. Wolff[2] states that when placed in Deetjen's culture-medium the **lymphocytes show undoubted changes of form,** which are **associated with slight locomotion.** These are, in his opinion, due to vital processes in the lymphocytes, and are not the mere effects of death of the cells and the like.

Gladin[3] describes some experiments that he has carried on concerning the **effects of leukotoxic serum.** He produced sterile skin-abscesses in dogs with aleuronat injections. The leukocytes were then washed 3 times with salt solution, and thus freed from serum. They were next injected into rabbits 8 or 9 times, at intervals of 8 to 10 days. The serum of these animals had marked leukotoxic effects *in vitro,* and only slight hemolytic action. By injecting it into the ear veins of dogs, the effect being controlled by the effect of the serum of an untreated rabbit, the author determined that the **leukotoxic serum causes a moderate temporary hypoleukocytosis and the appearance of nucleated red cells.** The erythrocytes regularly increase, except when fatal doses are given. A hyperleukocytosis follows the hypoleukocytosis. Even in fatal cases nucleated red cells are found. Megalocytes and megaloblasts were not observed. Gladin believes that the products of the leukocytes are factors that determine the wandering of the nucleated erythrocytes from the bone-marrow into the circulation.

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[1] Centralbl. f. innere Med., Aug. 24, 1901. [2] Berl. klin. Woch., Dec. 30, 1901.
[3] Allg. med. Centralzeit., Nos. 33 and 34, 1902.

S. Kaminer[1] presents a preliminary communication concerning his work on **toxinemia,** so called, his studies referring to the **iodin-reaction of the leukocytes.** A résumé of his results is as follows: The reaction is caused in rabbits and guinea-pigs by certain bacteria and their toxins. It is, for instance, produced by diphtheria toxin, but not by that of tetanus, and the production of a high degree of immunity to diphtheria toxin will interfere with the development of the reaction. He suggests that these differences between toxins in their relations to this reaction indicate differences in the action of the toxins, and perhaps especially biologic differences in the toxins themselves. Clinically, he states that the **reaction is always seen when toxins or bacteria are present** in the circulation in sufficient amount. The author considers the reaction of very marked diagnostic value, and of much importance in **furnishing indications for surgical procedures.**

In a discussion of Kaminer's paper, Lazarus[2] says that he has found the iodin-reaction of the leukocytes in numerous diseases. He does not believe that it is the evidence of a specific infection, but thinks that it **indicates infection in general.** Sometimes, however, either it is absent in infection, or but a trace is found. Lazarus believes that the reaction is due to the glycogen of the leukocytes. Michaelis agrees with him that the glycogen of the leukocytes is probably the substance which reacts to the iodin. Iodophilia, he believes, indicates degeneration. He agrees with Kaminer that **nonbacterial abscesses always show the absence of the iodin granules.**

Biffi[3] has studied the nature and significance of the iodophile granulations of the leukocytes, and believes that they are **not glycogen, but hemoglobin or a derivative of the latter.** He does not think that the iodin-reaction is sufficient to determine whether glycogen granules are ever present in the leukocytes or not. The presence of the iodophile granulations in the leukocytes he considers to be probably an evidence of phagocytosis. He believes that the iodophile granules, both intracellular and extracellular, correspond in all particulars to the eosinophile and pseudoeosinophile granules.

Galli[4] has studied the clinical value of the iodophile reaction of the leukocytes, and decides that, from this standpoint, the **reaction is of little or no importance.** He has found it in the most varied kinds of diseases and in normal persons, and the number of the granules has no relation to the intensity of any disease that may be present. As a rule, the reaction appears in association with a leukocytosis, but there is no constant relation between the intensity of the reaction and that of the leukocytosis.

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[1] Deut. med. Woch., Mar. 20, 1902.

[2] Centralbl. f. innere Med., No. 11, 1902, p. 281.

[3] Il Policlinico, No. 44, 1901.

[4] Il Policlinico, No. 44, 1901.

METHODS OF EXAMINATION OF THE BLOOD.

J. A. Capps[1] considers that valuable results are obtained by comparing the **percentage value of the blood-cells** as obtained in a graduated capillary tube, **by centrifugation, with the percentage number of the red corpuscles.** The quotient obtained he calls the **volume-index.** He has examined 14 cases of chlorosis, and finds a smaller diameter for the red cells, a decrease in the volume-index, and a more marked increase in the color-index. The diameter was low in 24 cases of secondary anemia; the color-index and volume-index were reduced, but less so than in chlorosis. In 17 cases of leukemia the conditions were much the same as in secondary anemia. Pernicious anemia showed a decided increase in the average diameter. The color-index was high; the volume-index still higher. Mild cases of chlorosis and secondary anemia and those that recovered showed a normal volume-index, even when the hemoglobin was still deficient. The chief **difference between chlorosis and secondary anemias,** from the standpoint of this method of examination, was apparently the tendency of the corpuscles in chlorosis to resist reduction in number more successfully than in secondary anemias. The enlarged size of the corpuscles in pernicious anemia the author considers an actual compensatory hypertrophy.

M. Einhorn and G. L. Laporte[2] describe a method for approximately **estimating the number of corpuscles from the stained specimen.** Comparing the results from this and from the usual method of counting, they conclude that it is a rapid and sufficiently accurate one.

H. Rosin and E. Bibergeil[3] contribute an article on **a study of the leukocyte in preparations which preserve its life for a short period.** They used neutral red, eosin, eosin-methylene-blue, pyronin-methyl-green, and magenta red methyl-green in staining. The most important of their observations they consider to be the following: (1) The demonstration of chromophoric zones, the sudden appearance of staining of the leukocytes, the nucleoli in the lymphocytes, and basophilic granulations in the erythrocytes of normal persons—in all these cases using methylene-blue. (2) The method of taking up neutral red and toluidin blue in ball-like form. (3) The spasmodic ameboid movements of the leukocytes and the marked movements of the granules before taking eosin and other acid stains. (4) The peculiar variability of the staining element of eosin-methylene-blue with white corpuscles, and the differential staining of the blood-plaques. (5) The existence of the red nuclear bodies in the blue nuclei of lymphocytes when pyronin-methyl-green is used; this is a characteristic of this form of cell, and the fine structure of their protoplasm is also notable. Further, the occurrence of an amphibolic stage of nuclear staining: the dying nucleus was an affinity for pyronin; the dead nucleus, for methyl-green. (6) The existence of a red structure in the chromatin of all blue nuclei when the magenta red methyl-green is used.

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[1] Chicago Med. Recorder, Aug., 1901.
[2] Med. News, April 19, 1902.
[3] Deut. med. Woch., Jan. 16 and 23, 1902.

R. May and L. Grünwald,[1] after directing attention to the fact that various preparations of eosin and methylene-blue give different results,—according as one uses distilled or tap-water in washing preparations, and as the water is used in a stream or the preparation is merely placed in standing water,—and also to the fact that the stains have a marked tendency to form precipitates and to change in their staining properties after being made up for some time, describe **a method of preparing a stain** which they consider superior to all others. They take 1 liter of 0.1 % eosin and the same amount of the same strength of medicinal methylene-blue, mix, and allow to stand for several days; then filter off the precipitate with a suction filter, wash until almost colorless, and use the dark precipitate as a stain—best in saturated solution in methyl alcohol. Preparations do not need to be fixed before being placed in this stain. They are spread on slides, allowed to dry, and immersed for about 2 minutes (in case the preparations stain badly, for a longer time; even as much as 24 hours) in the stain. They are then washed in distilled water which contains a few drops of the solution. Ordinary preparations are ready for the microscope 5 minutes after the blood is drawn. The distinction between the alpha and gamma granules is, the authors state, particularly clear; and the various elements of the cells are all clearly brought out. Precipitates are absent, and the method can be carried out with the greatest rapidity.

Tollens[2] discusses the **hemophotograph method of Gärtner,** and compares the results that they give in determining the hemoglobin with the Fleischl-Miescher hemoglobinometer ; 120 estimations, many of which were controlled by other persons, led him to the conclusion that the Gärtner instrument is useful for clinical purposes, if the costly hemoglobinometer is not at hand, and that the two give very similar results. The Gärtner instrument, however, has a **much larger error** connected with its use, and **requires more skill** for its manipulation.

CHLOROSIS.

Cavazza[3] discusses **urobilinuria in chlorosis.** After an examination of 20 cases, he decides that in acute chlorosis and in exacerbations of the disease one finds a marked increase of the urobilin, which, however, does not persist more than 4 to 6 days. Overstrain, cold, and hot baths may produce urobilinuria. As a result of these observations, the author concludes that either the **resistance of the red blood-corpuscles of chlorotics is reduced, or their blood does not preserve their normal isotonicity.**

Leclerc and Levet[4] contend, contrary to Gilbert, that **chlorosis is**

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[1] Centralbl. f. innere Med., Mar. 15, 1902.

[2] Centralbl. f. innere Med., June 21, 1902.

[3] Il Policlinico, Oct., 1901.

[4] Lyon méd., No. 31, 1901.

not essentially a disease of tuberculous and scrofulous persons. They find that two-thirds of the parents of chlorotic subjects show pathologic conditions, but these are by no means always tuberculous. In 42 cases they found neuropathic tendency 10 times, gout 7 times, tuberculosis 5 times, and diabetes, nephritis, chronic rheumatism, heart disease, and other conditions, each a smaller number of times. They found the blood-serum of chlorotic subjects more poisonous than is normal serum. They consider that 15 cc. per kilo is usually necessary as a fatal dose in rabbits. Serum from chlorotics was fatal in a dose as low as 8 cc. They think that chlorosis is **an autointoxication due to a disturbance of the intestines or to a disturbed ovarian function.**

A. Hofman[1] discusses the **rôle of iron in the production of blood.** From his experimental work, he decides that the iron ingested is absorbed from the duodenum, is then taken up by transport cells, in combination with albumin, and is deposited in the usual depots—the spleen, the liver, and particularly the bone-marrow. In the latter situation the iron may be found both in the cells of the bone-marrow and in the minute blood-channels. If animals that are made very anemic are given iron, the red blood-corpuscles are produced more rapidly, and the bone-marrow shows more myelocytes and a larger number of embryonal red cells than is the case in animals not so treated. The hemoglobin follows the course of the red cells, remaining somewhat behind in posthemorrhagic cases. It does not increase with especial rapidity in the animals given iron. The relative increase in hemoglobin in those animals is purely the result of the increase in the red blood-cells. The author considers that iron increases the number of red cells by increasing the rapidity of the production of mature red cells from embryonal cells; that is, **by stimulating the physiologic activity of the bone-marrow.** He states that it has **no direct effect upon the actual production of hemoglobin,** and believes that the purpose of iron is not merely to supply the iron lacking in the body. The **effectiveness of iron preparations is due to the amount of iron contained in them,** and the indications for different preparations of iron are not to be based upon their organic or inorganic nature, but upon the way in which they are borne by the patient. He describes chlorosis as a **disease due to reduction in the capacity of the blood-building organs**—a disease which appears about puberty and may disappear, or which is congenital and associated with hypoplasia of the blood-building organs, the circulatory organs, and the genital apparatus. Weakness of the blood-building organs shows itself in the production of erythrocytes which are of subnormal value, and which show abnormalities in form and in the amount of hemoglobin that they contain. The author believes that bleeding in chlorosis acts as a stimulant to the bone-marrow and is a rational treatment. If the body has lost its power of reacting to stimulation, iron is useless.

A. Jaquet,[2] in discussing the inorganic iron preparations, reaches the following conclusions: The organism has the power of repairing a deficiency in iron without especial regard to the nature of the iron preparation furnished; there is no **essential difference in the action of the inorganic and of the organic iron preparations,** and the choice of the preparation should not depend upon its organic or inorganic nature; organic preparations, however, seem to be better borne by the digestive tract, as a rule, and if inorganic iron is not well borne, the organic preparation should be tried; in most cases, however, the officinal iron preparations are wholly satisfactory.

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[1] Virchow's Archiv, Bd. CLX, Heft 2.

[2] Therap. Monatsh., No. 7, 1901.

PERNICIOUS ANEMIA.

Strauss and Rohnstein[1] discuss the blood-findings in 23 cases of pernicious anemia, making especial reference to the **condition of the leukocytes.** The average number of erythrocytes which they found was 1,240,000, but they note 2 cases in which over 4,000,000 were found. In 3 cases there were less than 500,000. Macrocytes were present in all the cases, and usually in large numbers. As to the leukocytes, the authors found that all but two cases showed a diminution, the average number being 5000; and they insist upon the **importance of a relative increase in the mononuclear cells,** as compared with the polymorphonuclear. The average of the former was 45.1 %, while that of the latter was but 52.5 %. An average of 37 % of the cells were lymphocytes, and the author states that in all cases the percentage of the polymorphonuclear leukocytes was below 70, while the mononuclear cells were regularly above 30 %. They consider that these observations are of importance in making a **diagnosis between pernicious anemia and gastric cancer;** for in the latter condition there is usually a tendency to leukocytosis, and the polymorphonuclear cells are increased, while the mononuclear cells are decreased. **Megaloblasts were absent upon repeated examinations** in 3 cases in which autopsy confirmed the diagnosis.

E. Bloch and H. Hirschfeld[2] consider that a relative increase of the lymphocytes is not an important diagnostic sign of pernicious anemia. They report a number of cases in which the **polymorphonuclear cells were increased decidedly** above the normal without any complications to explain this. They refer to cases collected from other authors showing the same condition.

T. McCrae[3] **discusses 40 cases of pernicious anemia.** He notes that but two of these cases were in negroes, while the average in the Johns Hopkins Hospital is 1 negro to every 7 white persons. The mouth was examined in 10 of these cases; **carious teeth or other septic conditions** were found in 4. Fourteen cases gave a previous history of malaria; 7, a family history of tuberculosis; 4, a history of syphilis; 3, one of mental distress and worry. In only one instance could a confinement be connected with the disease. The first symptom noted was in 14 cases weakness, in 9 dyspnea, and in 8 digestive disturbance. The author notes the frequency with which the patients complained of **loss of weight;** in 10 there was marked emaciation. The usual teaching is that there is no notable emaciation in this disease. Of the cases, 29 showed a typical lemon-yellow color of the skin; in 7 there was only pallor, without a yellow tint. **Pigmentation was present in 8.** The spleen could be felt in 6 cases, although the enlargement was never marked. General glandular enlargement was present in 3 cases, and there was considerable glandular enlargement in 5 other cases. The temperature was normal in only 10 cases and was more or less elevated in 27. A trace of albumin was present in 17 cases; sugar was regularly absent; 22 showed no gastric symptoms while under observation; 18 showed slight symptoms, 4 serious symptoms; 16 had normal bowel conditions; 14 were constipated; 8 had diarrhea, and 2 others, alternating diarrhea and constipation; 27 % showed nervous symptoms, these being chiefly sensory disturbances with a tendency to spasticity. There was an average of 37 erythroblasts in a count of 1000 leukocytes. About 23 of these were normoblasts, and 9.4 intermediate forms. The **average number of erythroblasts** in 11 fatal cases was 23 to 1000 leukocytes; in 16 cases that improved the average number of erythroblasts was only 7, and the average number of megaloblasts in these latter cases was less than one-seventh of the number in the fatal cases. The author finds different results from those of Strauss and Rohnstein concerning the leukocytes, the **multinuclear cells averaging 64%**, the mononuclear 34%, while the lymphocytes in the total number of cases averaged about 30 %. The last-mentioned authors, however, insist that incorrect figures will be obtained, unless the cells on both cover-glasses used in making the smear be counted. They find from 3 % to 6 % more lymphocytes on the upper glass.

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[1] Die Blutzusammensetzung bei den verschiedenen Anämien, Berlin, 1901.

[2] Berl. klin. Woch., Oct. 7, 1901.

[3] Jour. Am. Med. Assoc., Jan., 1902.

A. McPhedran,[1] in discussing the nature and treatment of pernicious anemia, refers to 22 cases that have been under his observation, and states that in 17 of these the condition of the mouth was noted, and that in **10 there were carious teeth,** but that in nearly all of these cases the teeth were too well cared for to be a possible source of infection. In only 4 were the gums unhealthy; and in 3 of these, only slightly so. There was no purulent secretion in any of these cases. One case showing grave symptoms of pernicious anemia had, 2 years before, shown dilation and prolapse of the stomach, without the evidences of pernicious anemia. The patient had recovered from the gastric condition before the onset of the pernicious anemia. In 2 cases there was a family history of what seemed undoubtedly to be pernicious anemia. **Symptoms of disease of the nervous system** occurred in 9 of the 22 cases observed. During 3 years, 14 have died; 4 are still showing a remittent course; the remaining 4 have passed from observation. One case apparently recovered entirely under arsenic; but McPhedran doubts the diagnosis. In **no other case has arsenic proved to be of any service.** Bichlorid of mercury, as a mouth-wash or internally, has been useless, as have other antiseptics. Normal saline has caused temporary improvement in some cases, and an apparent recovery in one case. Horse-serum was used subcutaneously in 4 cases, but caused only urticaria. Antidiphtheric serum was used in 2 cases with apparent improvement, in 1 case with no effect. Antistreptococcic serum in one case caused no good result. Spermin (Poehl) was followed by marked improvement in one case. Moderate purgation was used in several cases without any satisfactory effect.

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[1] Canad. Jour. of Med. and Surg., Dec., 1901.

A. Goodall[1] contributes a careful study of 9 cases of pernicious anemia, 6 in males and 3 in females. There were no satisfactory etiologic factors. The first symptom was usually marked weakness. Four had good teeth; in 4 the teeth were very bad; in 1 they were not carious, but were badly cared for. Splenic enlargement was present 4 times. There were heart-murmurs in all the cases. In 4 urobilin appeared occasionally in the urine. Nervous symptoms were found in 5 cases; 3 showed tenderness of the long bones. Five cases were fatal; 1 seemed to recover entirely. As to prognosis, age seemed to have no influence. Marked gastrointestinal symptoms, edema, and discoloration of the skin are not necessarily absolutely unfavorable signs. **Hemorrhages constitute a bad sign;** they occurred in 4 patients, all of whom died. Respiratory complications are bad. **Urobilinuria was observed in 4 of the 5 fatal cases, but in none of the others.** In the discussion of the conditions of the blood, the author notes that clot-formation is slow, even after marked improvement has occurred. **The lowest count of red cells was 295,000.** The author believes that a reduction below one million occurs only in pernicious anemia. If there is a tendency to reduction in the red cells observed within 2 weeks, the prognosis is probably bad; while a better prognosis may be established if there is a tendency to improvement within that period. Goodall considers the reduction of the white cells to be of importance in the diagnosis of secondary anemia, but finds the number of leukocytes somewhat higher than that usually given, his average being 5687. He states that with increase in temperature from any complication there is an increase in the number of the polymorphonuclear cells, either relative or absolute. The increase occurs largely at the cost of the lymphocytes.

Hamel[2] reports a **remarkable case of pernicious anemia** in a man of 42 years, in which the condition of the blood just before death was: hemoglobin, 10 %; red blood-cells, 275,000; white cells, 2400; specific gravity of the blood, 1.024; specific gravity of the serum, 1.021. The small lymphocytes constituted 51.9 % of the total white cells; the large lymphocytes were only 2.6 %; the large mononuclears, 1.8 %. There were 2.2 % of myelocytes. Postmortem showed chronic interstitial nephritis, chronic hepatitis, enlarged and indurated spleen, and dilation and hypertrophy of the heart; the gastric mucous membrane was not hypertrophied. The bone-marrow showed marked changes, and normoblasts and megaloblasts were present in about equal numbers in the bone-marrow; there were also numerous normocytes and megalocytes. Most of the white cells of the marrow were myelocytes; next to these came the small lymphocytes (about 25 %). The very low specific gravity of the blood has hardly been equaled in other reports. The **enormous number of megaloblasts**

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[1] Scottish M. and S. Jour., Nos. 1 to 4. 1902.

[2] Deut. med. Woch., April 17 and April 24, 1902.

in the circulating blood was striking. **To every 100,000 erythrocytes** were found, at several examinations, **42 normoblasts and 61 megaloblasts.** Most of the latter were so large as to deserve the name of gigantoblasts. Both megaloblasts and normoblasts had a striking nuclear ground-substance. Karyolytic changes were very common; basophilic granulation was present. The author particularly notes the extremely intense neutrophile staining of the granules of the polynuclear cells and the curious morphologic changes of the large lymphocytes, these cells show a strikingly intense staining of the narrow band of protoplasm, while the nucleus took an ill-defined, weak stain. The nucleus was oval and centrally situated. These were evidently degenerative changes. It was often impossible to distinguish satisfactorily between the megaloblasts and the large lymphocytes, because even their staining reactions were almost the same. The etiology of the case could not be satisfactorily determined.

O. K. Williamson and E. W. Martin[1] report an **interesting case of fatal anemia,** the blood-picture being in the main that of pernicious anemia. The reduction of the blood-cells progressed to 300,000 per cmm., while the white cells showed as many as 99.2 % of lymphocytes. The white cells were increased to as many as 45,000 per cmm.

Sawjalow[2] discusses the **chemical conditions of the blood** in a case of grave bothriocephalus anemia. The patient exhibited a reduction of the red cells to 2,100,000; the hemoglobin was 34 %. The treatment was the use of potassium salts and sweat-baths, and practically entire cure was obtained within 6 weeks. The relative amount of water in the blood was increased, the dry residue decreased, and the fat increased. These changes were decidedly more marked in the total blood than in the plasma, and the investigation of the solid elements of the blood showed a decided increase of fat and extractives. As a result of this conclusion, the author thinks that the **change in pernicious anemia is chiefly one of fatty degeneration.** He has attempted to find fat in the red blood-cells with osmic acid stain. The result was negative, but possibly this was because the patient was already nearly cured.

R. Bernert and K. v. Steyskal[3] contribute a study of the **minimal nitrogen metabolism in pernicious anemia.** They first give a very interesting discussion of the quantity of nitrogenous food absolutely demanded when the total caloric value of the food is kept at the normal point, and decide that the absolute minimum is about 0.48 gram of albumin (0.08 gram of nitrogen) per kilo of body-weight. The amount of this factor is independent of the ingestion of nitrogenous substances if the food has the proper caloric value. The demand for calories, also, may be largely reduced by gradually accustoming the individual to smaller amounts, particularly to smaller amounts of nitrogenous food. The authors' study of pernicious anemia from this standpoint was especially due to the claim made by Moraczewsky that there is, in all severe anemias, a reduction in metabolic processes, or what he terms "apathy and disassimilation weakness of the organism." Bernert and Steyskal find that the nitrogen minimum in pernicious anemia is no lower than in normal persons, and that therefore Moraczewsky has no basis for his claim. They say that their work showed definitely that in the two cases studied there was **no pathologic increase in nitrogen-metabolism.** This, however, does not indicate that the intoxication theory is positively incorrect, as the nitrogenous tissues might be shielded, under these circumstances, by marasmus or the relative obesity.

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[1] Brit. Med. Jour., May 10, 1902.
[2] St. Petersb. med. Woch., No. 27, 1901.
[3] Arch. f. exper. Path. u. Pharm., Bd. XLVIII, Hefte 1 u. 2.

E. Körmöczi,[1] in the discussion of a question as to the possibility of **diagnosing pernicious anemia from the appearance of the blood,** reports 5 cases of that disease in which the diagnosis was confirmed by a postmortem examination, and especially dwells upon the fact that in 3 of these cases **nucleated red blood-cells could not be found in the circulating blood,** and that in 2 of them the blood-corpuscles were of normal type. In the third case there were numerous macrocytes, and in the other 2 nucleated cells, some of them megaloblasts, were discovered. In all the cases the bone-marrow contained nucleated cells, many of them being megaloblasts. This is evidence that the absence of megaloblasts, or of nucleated red cells from the circulating blood in general, is not sufficient proof that pernicious anemia is not present; the previous reports in recent literature have satisfactorily demonstrated that the **presence of megaloblasts does not establish the diagnosis** of that disease. The author considers, however, that their presence is **sufficient for a tentative diagnosis** of pernicious anemia, in the absence of signs of other conditions. He has made some attempt to learn why megaloblasts or nucleated cells are present in the circulating blood at times and absent at other times, when the bone-marrow probably contains nucleated red cells in each case. He particularly notes that he administered various substances that have a marked chemotactic action, but was unable, by this means, to cause nucleated cells to appear in the circulation. The nucleated red cells, however, did seem to appear coincidently with myelocytes and to disappear with these cells.

LEUKEMIA.

Löwit[2] contributes a general review of his previous work concerning **parasites in leukemia** and reiterates his views concerning this question.

Löwit[3] recommends that **in looking for the hæmamœba** a moderately thin film should be prepared by drawing blood across a cover-glass with a platinum needle. The preparations are fixed by heat; stained with concentrated thionin, which is warmed until it steams slightly; washed, after cooling, in water; dried in air; then stained in diluted Lugol's solution for 10 to 20 seconds; rewashed; and mounted in balsam. By this method the parasites appear a dark bluish-black, the leukocytes are light blue, and the erythrocytes are unstained. The smaller intracellular forms are, however, often overlooked by this method, as they are frequently obscured by the general coloring of the leukocytes or by the basophile granules. For this form the author recommends, instead of using the iodin in the above method, counterstaining with lithium-carmin solution (Orth) for from 1 to 10 minutes. He believes that this **acts as a specific stain,** and demonstrates that **the parasites have a very constant form and are of two kinds:** the round variety, usually found in the leukocytes; and the crescent, spindle, or half-moon form, generally extracellular. The last form have flagella, and, therefore, probably have motion; and Löwit thinks that this is the first form in a developmental cycle, which probably does not reach its end in man. The round form probably develops through sporulation; and ring forms appear, which usually have a vacuole in the center, and sometimes have a nucleated inner body. As a rule, the infection is a simple one. Sometimes the different varieties of parasites are seen in the same specimen of blood, and perhaps in the same leukocyte. The large round form is the most common.

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[1] Deut. med. Woch., Jan. 2, 1902. [2] Zeitschr. f. Heilk., Bd. xxii, 1901.
[3] Zeitschr. f. Heilk., 1901, Heft 7.

H. Hirschfeld and E. Tobias[1] report 2 cases of myelogenous leukemia, with **extensive differential counts and careful studies of the staining reactions of the leukocytes.** They note that in both cases there was pronounced polymorphism in the leukocytes. In both, the large and small mononuclears, the mast-cells, and the eosinophiles showed most remarkable changes in number. Most of these varieties of cells were at times absent. In both cases, also, the staining reactions were striking, more particularly the staining reactions of the granules. The myelocytes at times contained extremely small numbers of granules, and these in part took a distinctly bluish tinge with either the triacid or the methylene-blue mixture. A similar staining was observed in the granules of the polymorphonuclear leukocytes, and the **eosinophiles at times showed both red and blue granules.** At times, also, the granules of these cells stained a pure blue. It has previously been noted that neutrophile granules, in the earlier periods of the existence of the cells, may take basic stains. In one case it was noted that the **polymorphonuclear cells contained no granules**—a condition which has previously been described, and which apparently indicates a lack of power to reproduce the neutrophile substance. In the same case the difference in the size of the leukocytes was extremely striking, some being giant-like cells, and some being unusually small polymorphonuclear cells. The mast-cells in this case showed decided changes in the staining reactions of the granules. This case was complicated with miliary tuberculosis, and it is noted that the leukocytes fell from over 400,000 to about 280,000 at the time of death, the red cells in this case having decreased from about 3,000,000 to about 1,900,000. The authors briefly describe the 10 cases of **leukemia associated with tuberculosis** that have been reported. These cases are of two varieties: those in which tuberculosis existed in latent form and was set aflame by the leukemia; and those in which tuberculous infection occurred during leukemia, because of the lessened resistance of the individual. In one case a tumor of the ear was found to be a leukemic infiltration of a fibroangioma. The authors looked carefully for Löwit's protozoa and found similar bodies, but considered them to be degeneration forms. They noted that their number went hand in hand with that of the mast-cells. They injected the juice of the spleen, lymph-glands, and bone-marrow into rabbits, without producing pathologic conditions other than embolism and sepsis.

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[1] Deut. med. Woch., Feb. 6, 1902.

Sturmdorf[1] reports a case of combined **leukemia and tuberculosis,** the patient when first examined showing evidences of pulmonary tuberculosis with coincident laryngeal involvement. He especially directs attention to **changes in the proportion of the leukocytes** during repeated febrile attacks. During the fever the myelocytes decidedly decreased, while the polymorphonuclear neutrophiles increased markedly, the total number of the white cells and the percentage of the other cells remaining about the same as before. The author believes that the increase of the polymorphonuclear cells in the febrile period indicates an **intercurrent leukocytosis.**

G. Parker[2] reports a case of **splenic leukemia** which occurred in a man **with old phthisis.** The tuberculosis of the lung became active during the course of the leukemia. During this time the percentage of polymorphonuclear cells decreased markedly, while the myelocytes increased from 14 % to 25 %.

E. Schwarz[3] reports a case of **leukemia with giant-cell embolism and osteosclerosis.** The patient was 47 years old, and had exhibited very marked splenic tumor and 40,000 leukocytes, the number of the latter decreasing shortly before death. The red cells had shown little change. There were numerous neutrophile myelocytes and erythrocytes. The mononuclear elements were both absolutely and relatively markedly increased, as were the eosinophiles. The necropsy showed splenic tumor, enlargement of some lymph-glands, hyperplasia of the bone-marrow, with osteosclerosis of the spongiosa and of the diaphysis. The author considers this an early case of leukemia. There were curious objects, very rich in chromatin, found in the capillaries during life, and these he considers to be **degenerated nuclei of bone-marrow giant-cells.** There were found in the capillaries of the liver, kidney, and spleen, at postmortem, masses of chromatin, which were apparently identical with these bone-marrow giant-cells; and the giant-cells were enormously increased in the marrow itself. Schwarz made some **attempts to produce giant-cell embolism** in rabbits by the methods of Lubarsch and Lengemann, but had a positive result in only 1 of 7 attempts; in this he injected the liver-emulsion of a rabbit into the peritoneal cavity of another rabbit. He thinks that such embolisms are produced by chemotropic action, and believes that their occurrence is strong proof of the myelogenous theory of leukemia. He notes that in this case the osteosclerosis was very marked, while the leukemic changes of the bone-marrow were merely beginning; hence, the osteosclerosis was merely the result of hyperplasia of the bone-marrow.

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[1] Am. Jour. Med. Sci., Aug., 1901.

[2] Brit. Med. Jour., May 10, 1902.

[3] Zeitschr. f. Heilk., 1901, Heft 11.

E. Adler[1] discusses 23 cases of leukemia observed in v. Jaksch's clinic. Two of these showed a **sudden marked reduction of the leukocytes**—the one as the result of an intercurrent sepsis, the other without discoverable cause. In the one case—that of sepsis—the mononuclear neutrophiles and the eosinophiles showed very pronounced reduction, while the lymphocytes at the same time showed an increase, This was not observed in the other case. It is striking, in both cases, that there was an unusually marked number of typical mononuclear eosinophiles.

M. Bundi[2] reports the **clinical and anatomic changes in the eye-grounds** in a case of lienal leukemia, in which he reaches the conclusion that the changes in the retina are not inflammatory, and that, consequently, the expression "leukemic retinitis" is an improper one. The process begins in a vascular change.

ACUTE LEUKEMIA.

Hirschfeld and Alexander[3] describe a remarkable case of **acute, apparently myeloid leukemia,** which occurred in a man of 21, who had previously been in good health. He first exhibited anemia, then fever, enlargement of the spleen, pallor, and various signs of leukemia, in the last 2 weeks the course of the disease being very rapid and associated with marked prostration. Upon examination the hemoglobin was 35 %; the red cells, 2,000,000; the white cells, practically normal. Four weeks later the hemoglobin was 15 %; the red cells, 1,250,000; the white cells, 30,800. There were marked alterations in the size of the reds, and normoblasts were present. The leukocytes showed a marked relative increase in the lymphatic elements—both the large and the small lymphocytes. The myelocytes also were increased. There was sternal pain, multiple periostitis, leukemic tumors of the mucous membrane of the mouth, and leukemic retinitis. The postmortem showed enlargement of the cervical, retroperitoneal, and mesenteric glands, and of Peyer's patches; and a very marked splenic tumor. The red bone-marrow contained only a few nucleated or nonnucleated red corpuscles, and chiefly consisted of granulated white corpuscles, which were large myelocytes; the white marrow had the same appearance. Upon microscopic examination the spleen and lymphatic glands were found to show extreme myeloid infiltration, and the growths of the mucous membranes showed the same appearance. Neither eosinophiles nor mast-cells were found in the circulating blood. Nevertheless, the authors **think that the case was a myelogenous leukemia,** and suggest that the **injury of the bone-marrow was so severe** in this case that it is possible that the newly formed eosinophile cells were at once destroyed.

D. D. Stewart[4] reports a case of acute leukemia that occurred in a girl of 28. When admitted, the patient was pallid; she had a tendency to nose-bleed; **her gums were swollen and bled readily;** and her teeth were loose. She had slight temperature, which rather decreased than increased for a week. This was then followed by a rise and by a **course resembling typhoid fever.** About 4 weeks after admission an examination of the blood showed 89,000 leukocytes, 1,280,000 red cells, and 25 % of hemoglobin. The leukocytes increased decidedly after this, and the patient died a month after admission, following large hemorrhages from the bowel. The postmortem examination showed a general leukemic infiltration of the tissues, with no lesion of typhoid fever. The blood-picture was that of the large-cell type.

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[1] Zeitschr. f. Heilk., 1901, Hefte 8 u. 9.
[2] Prag. med. Woch., 1901, No. 26.
[3] Berl. klin. Woch., No. 11, 1902.
[4] Am. Jour. Med. Sci., Nov., 1901.

Mannaberg[1] reported to the Society for Internal Medicine in Vienna a case of acute leukemia in a patient nineteen years of age. Because of the presence of **a maculopapular syphilitic eruption,** an injection of 2 cg. of corrosive sublimate was given. The patient soon showed severe weakness. Fever began after a week. After another week there were severe **stomatitis and epistaxis,** and the general picture was that of foot-and-mouth disease. Later the patient had rigidity of the neck, swelling of the spleen and of the submaxillary glands, and marked increase in the number of the white cells of the blood—62 % **being polymorphonuclear,** 14 % large lymphocytes, and 24 % small lymphocytes. The blood-picture spoke for a combination of leukemia with sepsis. The autopsy showed gangrenous ulceration of the pharynx and multiple embolic abscesses in the kidneys. The intestines showed no evidence of corrosive-sublimate poisoning. Spieger, in the discussion, suggested the **possibility that the original eruption was due to leukemia.** Berdach noted that the blood-picture was not typical of leukemia, and **thought it possible that sepsis explained the whole condition.**

M. Mixa[2] reports a case of acute leukemia which **began with severe hemorrhage from the gums.** There was no general purpura and no general enlargement of the glands. Fever appeared during the last few days of life. The course was 10 weeks in length and ended with cerebral hemorrhage. The lymphocytes were increased to 93 % of the total of the white cells; eosinophiles were absent.

G. W. Wende[3] reports a case of **Hodgkin's disease** in which the first abnormal development of lymphoid tissue was in the skin, and in which there were subcutaneous and internal hemorrhages and spontaneous increase and reduction of the tumors. There was then a **suddenly developing change in the clinical picture,** and the blood showed alterations characteristic of lymphatic leukemia, in that the red cells became very much reduced, while the white cells rose from 4000 to as high as 45,000; the small lymphocytes increased to as high as 95 %, although the differential count had previously been normal. There was a terminal tonsillar infection, which resulted in a general sepsis, and this was accompanied by a subsidence of the lymph-nodes and of the splenic tumor, and by a reduction of the leukocytes to 600 per cmm. The small lymphocytes were still present in an abnormally large percentage. Inoculations and bacteriologic studies were negative.

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[1] Centralbl. f. innere Med., Mar. 1, 1902, p. 234.

[2] Wien. klin. Rundschau, No. 37, 1901.

[3] Am. Jour. Med. Sci., Dec., 1901.

LEUKEMIA-LIKE DISEASES.

R. v. Jaksch[1] gives a further discussion of a case previously reported, in which there were multiple involvements of the periosteum in **a case closely resembling myelogenous leukemia.** In the further course of the case the changes in the periosteum grew much more marked. There was a marked increase in the leukocytes, particularly in the mononuclear neutrophiles; less marked in the mononuclear eosinophiles. There is an extensive report of the postmortem findings, and a discussion of the changes. These lead the author to a restatement of his previously published conclusion, that it was not a case of leukemia with previously unobserved changes in the bone, but an actual new disease-picture, which he calls **multiple periostitis with myelocythemia,** the cardinal symptoms being multiple ostitis, enlargement of the spleen, a peculiar blood-picture,—viz., myelocythemia with reduction of the red cells,—pains which afterward disappeared, persistent sweats, and the hemorrhagic diathesis.

L. Michaelis[2] reports a case belonging to the group of leukemia-like diseases, which occurred in a woman of 50. She exhibited a **disease which produced death within 3 months.** She showed marked enlargement of the spleen, which remained soft, no swelling of the lymphatic glands, a temporary appearance of normoblasts in the circulating blood, and **no marked increase in the total number of the leukocytes,** the bone-marrow being found red postmortem. The total leukocyte count was, at highest, 16,000. The **lymphocytes constituted 76 %**; the myelocytes, 7.2 %; and the eosinophiles and mast-cells, each only 0.4 %. The bone-marrow consisted almost entirely of lymphocytes and giant-cells, with a few myelocytes. The spleen showed a similar appearance, and in various organs bone-marrow giant-cells were found. If Pinkus's classification be accepted, the case conforms most closely to the type of pseudoleukemia, as this author's definition of pseudoleukemia practically includes lymphatic leukemia; but it differed from this type in having an **absence of swelling of the lymph-glands,** and, more particularly, in the **presence of signs of marked activity of the bone-marrow;** also, the hemorrhagic diathesis was absent. The difficulty in the classification of cases of this kind would, the author thinks, be much reduced if the cells included among the lymphocytes were more carefully differentiated, and, more particularly, if the classification suggested by himself were adopted—that is, especially if the large lymphocytes without granulations were called **lymphoid cells,** with the idea that they are very possibly **undifferentiated connective-tissue cells** from lymphoid tissue, which may subsequently become granulated cells. These cells then become merely an indication of the return to the embryonal condition of blood-production or the persistence of an embryonal type of blood-production.

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[1] Zeitschr. f. Heilk., 1901, Hefte 8 u. 9.

[2] Zeitschr. f. klin. Med., Bd. XLV, Hefte 1 u. 2.

J. A. Scott[1] reports the case of a **child 9 months old** when first seen, with a good family history, except that a sister, 12 years old, was said to have had a large spleen and to have been anemic since birth. The patient was well until he was a month old. Then he showed pallor and had recurring intestinal disturbance; and when first seen the spleen was much enlarged. There were no signs of rickets. The liver and lymphatic glands were not enlarged. Spreads at this time showed decided leukocytosis, with a large proportion of lymphocytes and irregularity in the red cells. The intestinal disturbance continued, and neuroretinitis developed; some blood was expectorated, but there was no other hemorrhage. The liver became palpable about a year after the child was first seen, and death occurred soon after this. The red cells varied very little, while the leukocyte count varied from 105,000 to 18,800. The lowest percentage of mononuclears was 44; the highest, found just before death, was 99. Myelocytes were present. The blood-picture **corresponded with the description usually given of v. Jaksch's disease,** but Scott could not find differential counts made in this condition. He believes that the condition closely corresponds, in its various features, with the leukemia of the adult.

Treatment.—M. Franke[2] describes a case of lymphatic leukemia in which he had glands from the axilla extirpated and **used an extract of these glands for injections into the peritoneal cavity of rabbits,** subsequently killing these animals and determining the **effect of their blood-serum upon the blood of the patient.** He states that when examined under the microscope, the effect was seen to be chiefly upon the small forms of the lymphocytes, while the large varieties were affected much more slowly and much less markedly. The changes consisted in increased transparency of the cells, irregularity in their outline, and, finally, a shadowy appearance or an entire disappearance of the cells, with the exception of a finely granular mass. These changes occurred within from 6 to 8 hours. They were not seen when the rabbit serum was not added, nor when the serum of a normal rabbit was used. They were attributed to **leukolytic qualities of the serum of the injected rabbits.** It was intended to treat the patient with this leukolytic serum, but his condition was so bad as to prevent this undertaking. The author also states that **he intends to try the same procedure with sarcoma.** In the case reported the small mononuclear leukocytes constituted 78 %; the large mononuclears, 17 %; the polynuclear neutrophiles, 4 %; and the transitional forms, 1 %. [The treatment of conditions associated with a high increase of leukocytosis by any substance which has a marked leukolytic effect might be highly dangerous because of the danger of intravascular thrombosis, and should be well studied in animals before being used in human subjects.]

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[1] Am. Jour. Med. Sci., Jan., 1902.

[2] Centralbl. f. innere Med., Feb. 8, 1902.

HODGKIN'S DISEASE.

E. Becker[1] discusses **lymphomas from a clinical and pathologic standpoint,** basing his paper upon the following case. A woman of 33, who had previously been in good health, and whose family history was entirely negative, developed swelling of the lymphatic glands in the neck. This swelling gradually involved other glands, and within 3 years practically all the superficial glands were swollen, some of them to the size of a walnut. The spleen was moderately enlarged; there was decided relative increase in the leukocytes, particularly of the large form, but only slight increase of the general leukocytic count. (The highest count was 11,200.) All the other organs were normal. The tumors were evidently of benign character. The general health was very slightly affected, and treatment by injections of arsenic caused the glandular swellings and the enlargement of the spleen to decrease considerably. He first divides his discussion of lymphomas under the following heads: Lymphosarcoma; malignant lymphoma of Billroth; multiple tuberculous glandular enlargements; luetic lymphoma; anæmia splenica infantum, and essential pseudoleukemia. Discussing these various forms, however, he thinks that there is **no decided distinction between the malignant lymphoma of Billroth and pseudoleukemia.** There is no very good distinctive point between those cases which are of nonmalignant course and those which run the distinctly malignant course described by Billroth. Since, then, the general characteristic of all these classes which have been mentioned consists in the lymphatic swelling, Becker considers it best to divide the cases under the following **5 headings,** instead of 6: (1) Lymphosarcomatosis; (2) tuberculous lymphomatosis; (3) luetic lymphomatosis; (4) simple hyperplastic lymphomatosis, with the subheading anæmia splenica infantum; (5) diffuse lymphomatosis, the latter including pseudoleukemia and malignant lymphoma. He believes that **pseudoleukemia and lymphatic leukemia are due to the same cause,** and that the only difference in the two diseases is the presence of a larger or smaller number of lymphocytes in the blood; this difference is often merely a secondary one, the cause of which is not yet distinctly known. He believes that the **term "pseudoleukemia" should be abandoned,** and that **"diffuse lymphomatosis" is a better term.** One can attach the subheading "leukemica" or "aleukemica," acute or chronic, lienal, adenoid, myeloid, etc., as circumstances indicate.

H. Schür[2] demonstrated to the Vienna Medical Society blood-preparations from a case of **universal lymphomatosis** previously demonstrated to the same society. Necropsy had shown tuberculosis, involving the superficial lymph-glands and the spleen, with numerous chronic tubercles in the latter organ. There were also nodules in the lungs, a nodule in the right ureter, which caused hydronephrosis in this side, and amyloid disease of the liver, spleen, and kidneys. Microscopically the cells especially noted by Sternberg as characteristic were found in considerable numbers. **Tubercle bacilli were also present.** The tonsils were found normal. The bronchial lymph-glands were swollen. The pharyngeal tonsil was not examined. The exact point of entrance could not be determined. Türk, in the discussion, **considered the designation tuberculous pseudoleukemia unfortunate.** He believes that there is a decided difference between the cases of true hyperplastic lymphomatosis and chronic tuberculosis of the lymph-glands, both in the blood-conditions and in the clinical symptoms. The occurrence of marked fever, particularly of a hectic type, and the tenderness of the glands,—both of which symptoms were present in Schür's case,—and the absence of diffuse or localized nodules due to swelling of the lymphatic apparatus of the mucous membranes, particularly those of the mouth and throat, usually distinguish the tuberculous from the true hyperplastic form. Sternberg, in the discussion, also stated that the name tuberculous pseudoleukemia is a bad one. He likewise had noted the tendency to the occurrence of febrile movements in the tuberculous condition, and insisted upon the regular appearance of the disease elsewhere than in the lymph-glands. The mononuclear leukocytosis, which was mentioned as of such importance, may appear in a great variety of diseases of the lymphatic apparatus, and may also be absent. Türk agreed with the view concerning lymphomatosis; while Schür considered the absence of a relative or absolute lymphomatosis very important for the diagnosis of the tuberculous condition, and believed fever and tenderness of the glands to be of no importance in diagnosis.

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[1] Deut. med. Woch., Oct. 17 and 24, 1901.

[2] Centralbl. f. innere Med., April 19, 1902, p. 412.

H. B. Shaw[1] reports a case of **lymphadenoma with periodic attacks of fever,** which were accompanied by marked constitutional disturbances, particularly gastrointestinal disturbance and chills, these general signs decreasing as the fever disappeared. At times, also, the skin became reddened over some of the packets of glands. The author also gives a table of 17 similar cases collected from the literature, in which there was terminal recurrent fever; and discusses the theories of causation of this disease.

C. F. Martin[2] discusses a case in which there were some **previously undescribed lesions in the lymphosarcomatous form of Hodgkin's disease.** The case was that of a man of 55, who had general enlargement of the lymph-glands, followed by exudates in the pleural and peritoneal cavities, the fluid being repeatedly removed. It was of a rather milky character, became clear after shaking with ether, and contained many leukocytes and fatty cells. Autopsy showed that the serous membranes, particularly the peritoneum, were covered with **thick masses of granular or cauliflower-like new-growths.** These could readily be torn off; and beneath, the peritoneum seemed smooth and uninflamed. Microscopically the growths were composed of lymph-cells, with some spindle-cells and with a fine stroma. The case had exhibited the clinical picture of pseudoleukemia, and undoubtedly belonged in the class of lymphosarcomatosis.

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[1] Edinb. Med. Jour., Dec., 1901. [2] Jour. of Med. Research, 1901, No. 6.

W. Cottle and L. Dickinson[1] report 3 cases of Hodgkin's disease in which they observed marked **pruritus,** a condition which has repeatedly been described in this disease. Two of the cases ran a very rapid course. The patients did not present any evident skin-lesions. The cause of the pruritus may be either toxic influence or poor nutrition of the skin.

THE HEMORRHAGIC DISEASES.

SCURVY.

Lamb[2] discusses a series of 11 cases of scurvy, with especial reference to the **theories of acid intoxication, of ptomain-poisoning from eating tainted meat, and that of Liston,** who claims that it is due to Ankylostoma duodenale. Five cases developed in a jail, in which the diet had been exactly the same as that given to 1095 other prisoners. The meat was eaten the day it was killed, and was given only once a week. The author believes that this **excludes the ptomain theory,** and also Wright's theory of the dietary containing an excess of animal acids. The **alkalinity of the blood was also determined and found normal,** and the **stools were examined for ova with negative results.** The symptoms did not improve upon the use of food-stuffs rich in alkali, or upon the administration of large doses of sodium lactate. The 6 other cases were from outside; and the diet, etc., previous to admission could not be learned. The conditions while under observation, however, **gave no support to any of the theories mentioned.** In 2 cases ova of parasites were found in the stools: in one case an ovum of Trichocephalus dispar; in the other, ova of Trichocephalus dispar, Ascaris lumbricoides, and Dochmius duodenalis. Lamb believes that it is possible that acid intoxication may be a factor in scurvy, but he considers that the condition clinically known as scurvy is undoubtedly due to more than one etiologic factor, and is associated with more than one pathologic condition.

Müller[3] found in the urine of **2 out of 3 cases of scurvy an organism** that closely resembled the one described by Babes, in 1893, as having been cultivated from the gums. The author does not definitely decide whether the bacillus has any causal relation to scurvy. Cultures injected into a rabbit produced no results.

Michelazzi[4] reports a case of scurvy in which he believes that **intestinal autointoxication** existed and was the cause of the disease. He considers the latter condition to be the cause of many cases of scurvy.

PURPURA.

G. Carriere[5] reports a case of purpura which occurred in a boy of 14. The purpuric eruption was preceded for several days by rheumatic pains and gastrointestinal disturbance; then, without any fever, there appeared a widespread purpura, which involved a large part of the skin surface, including the face and also the mucous membrane of the mouth. The marked gingivitis suggested scurvy, but the absence of deep hemorrhages and of asthenia seemed to exclude this. There was a marked leukocytosis and a decided hemophilia. **Pure cultures of the bacillus described by Achalme** were obtained. The author believes that this case indicates that the **old theory that purpura is sometimes rheumatic in origin is probably true.**

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[1] Brit. Med. Jour., Aug. 24, 1901.

[2] Lancet, Jan. 4, 1902.

[3] Münch. med. Woch., Nov. 12, 1901.

[4] Gaz. degli osped., 1901, No. 114.

[5] Arch. de Méd. exper., p. 149, 1901.

Cohen[1] describes a case of **associated phthisis and purpura.** The patient, a girl of 19, noticed a blister-like eruption 10 days before admission. This was painful. The legs became edematous. On admission there was slight glandular enlargement. The joints were free, the legs were edematous, and there was a purpuric eruption on the legs. The lungs were then normal. Blood-cultures were negative. The heart was somewhat enlarged. She afterward vomited blood, and a new eruption appeared. Subsequently she developed severe abdominal pain with signs of shock. There were repeated eruptions on the skin and mucous membranes. Bloody stools were passed. Hemorrhagic nephritis appeared, and the signs of tuberculosis of the lung became evident. Autopsy showed advanced pulmonary phthisis, no cardiac abnormality, no tuberculosis of the peritoneum, a nutmeg liver, fatty degeneration of the kidney with numerous hemorrhages, and enlargement of Peyer's patches.

Pratt[2] reports a case of purpura **associated with tuberculosis.** Lung signs were absent at first. The patient had nose-bleed and some fever, afterward hematuria, and then signs of involvement of the lungs appeared. There were numerous scattered hemorrhages, and there was suppuration in the parotids. At the time of death the whole body was covered with purpuric spots. Autopsy showed widespread tuberculosis. The condition of the heart is not noted.

Underwood[3] reports a case of **pneumococcic infection, with purpura,** in a boy 4½ years of age. When first seen, on February 7th, lobar pneumonia was present. There was an erythematous rash. The crisis occurred on the tenth day. The patient did well until March 1st, when he had epistaxis and hemorrhage from the bowel, and there were purpuric spots under the skin and mucous membranes. The hemorrhage continued intermittently. Fever developed on March 11th. There were cough, sweating, and hemoptysis for 2 days, and some oozing from the gums. The hemorrhage ceased after this, and the child completely recovered. The epidermis exfoliated in large plaques. The author found in blood-smears numerous **colorless, highly refractive granules,** which, he believes, were identical with the blood-dust of Müller. He thinks that these granules are derived both from the nuclei of red cells and from leukocytes.

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[1] Münch. med. Woch., No. 50, 1901. [2] Brit. Med. Jour., Sept. 28, 1901.
[3] Amer. Med., Oct. 19, 1901.

HEMOPHILIA.

Holtschmidt[1] reports 5 cases of hemorrhage in hemophiliacs treated by **injection of 15 cc. of 2 % gelatin solution,** half of the quantity being given in one place and half in another, to avoid too great a tension on the tissues. He considers the results to be demonstrative of the favorable influence of the gelatin.

Damianos[2] reports a case of hemophilia in a man of 23, in which severe hemorrhage **followed the removal of a tonsil,** requiring compression and finally ligation of the carotid artery. The patient, however, died. An injection of 2 % gelatin had been made during the course of the bleeding, and at the point of injection a **gas-containing abscess, evidently due to infection of the gelatin,** had formed.

C. E. Wallis[3] reports 2 cases of hemorrhagic diathesis in which he used **calcium chlorid** before removing a number of teeth. In neither case did excessive hemorrhage occur. One patient had some hemorrhage during a brief period when she did not take the drug as ordered, but it stopped after resuming the calcium chlorid.

DISEASES OF THE SPLEEN.

A. Bentivegnia,[4] in a contribution concerning the **physiology of the spleen,** reports his observations after the removal of that organ. He finds that the toxicity of the blood increases during the first few days after the operation, as does that of the urine. When animals from which the spleen had been removed were compared with control normal animals, it was found that the injection of the colon bacterium, the use of spoiled meat, and the production of an artificial occlusion of the intestine produced more marked changes in the liver and kidney of these than in the same organs of the control animals. The author considers that this demonstrates an **antitoxic function** of the spleen, and thinks that the disease of the liver in primary splenomegaly ("Banti's disease") is explained in this way.

Wentworth[5] contributes a very careful discussion of the subject of **anemia with chronic enlargement of the spleen.** He concludes that the **blood-changes in so-called "splenic anemia" are those of secondary anemia,** and that the degree of cachexia which has been described in these cases does not usually correspond with the blood-changes, the anemia being more moderate than the cachexia. This makes it more probable that the anemia and the splenic enlargement are the results of a primary condition which produces both. The **source of intoxication is probably manifold.** The changes in the spleen are almost purely overgrowth of fibrous tissue. The latter itself cannot produce intoxication; and while it may interfere with the function of the spleen, the functions of that organ are not essential to life or, apparently, to health. Hence, the overgrowth of **fibrous tissue observed in the spleen could not be the cause of the symptoms.** The splenic functions are not in any sense analogous to those of the thyroid and suprarenal glands. Even if the changes in the spleen were such as to produce intoxication, it is impossible to account for the absence of such intoxication in the exactly similar chronic hyperplasia of the spleen, which is associated with a variety of well-known causes. It has not been proved that the splenic changes are primary; and, indeed, it is probable that they are not, both from the pathologic characteristics and from clinical observation in a number of cases. The lesions found in the spleen are not those of pseudoleukemia. The author thinks that there has been too much of a tendency to generalize from observations made on one or two cases; that **in so-called "splenic anemia" nothing characteristic of a primary disease** has been discovered in any organ or in the blood; and that there is almost unquestionably a diverse etiology. The pseudoleukemic disease of infants is, he thinks, unquestionably a secondary anemia, the peculiar appearances being due to the age of the subjects. This condition is also identical with infectious splenic anemia, and there is no real proof that the anemia splenica infectiva of the Italians is a primary disease of the spleen. There does not seem to be any apparent relation between the character of the blood and the changes in the spleen in infancy; similar lesions may be found in the spleen with severe anemia or with practically no anemia, and with extremely varying conditions of the leukocytes. The duration of the condition does not explain these differences; and the author believes that we are justified only in deciding that in secondary anemia chronic hyperplasia of the spleen sometimes occurs and at other times does not. He considers the names "splenic anemia," "anæmia splenica infectiva," and "anæmia infantum pseudoleukemica" to be objectionable, and thinks that there is no evidence to justify the use of the word "primary" in connection with splenomegaly, or the attaching of the term "infectious" to the infantile form of splenic anemia.

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[1] Münch. med. Woch., Jan. 7, 1902.
[2] Wien. klin. Woch., No. 9, 1902.
[3] Brit. Med. Jour., May 10, 1902.
[4] Lo Sperimentale, 1901, Fasc. 4.
[5] Boston M. and S. Jour., Oct. 3 to 31, 1901.

J. Ullman[1] describes the case of an Italian of 49, who had had malaria 5 years before. The liver was small, the spleen enormously enlarged. There was a trace of bile in the urine. The glands were not enlarged. The red cells were 3,532,000; the whites, 4400; and the hemoglobin, 40 %. There were no plasmodia of malaria. The case was considered to be one of **splenic anemia in a malarial subject.**

B. Robinson[2] presented a case to the Practitioners' Society to show the **results of surgical operation in splenic anemia.** Enlargement of the spleen, which was observed when the patient was 14 years old, was then thought to be due to malarial poisoning, but the presence of malaria could never be demonstrated. He had also slight anemia. The leukocytes were 3125 per cmm. The differential count was normal. The spleen was removed by Markoe. Following the operation the leukocytes increased to 17,000, and there was a relative increase in eosinophiles. When presented, 8 years after the operation, the boy was in good health. [It is a serious question, if operation is to be considered, where the limits of the term splenic anemia shall be placed. This case is here reviewed from a brief society report, and we do not wish to offer criticism of the attendant. But it must be remembered, in general, that mere splenic enlargement and slight blood-changes do not constitute a disease or justify a dangerous operation.]

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[1] Buffalo Med. Jour., July, 1901.

[2] Med. Rec., Jan. 8, 1902, p. 113.

S. Sawelgaw[1] describes a case of **wandering spleen** which exhibited chiefly the **signs of gastroenteritis and marked pain** in the region of the umbilicus. The spleen could not be determined to be in its normal position, and a tumor corresponding in size to an enlarged spleen was found in the umbilical region. The child had had smallpox a short time before, and had also twice fallen. This history seemed to explain the enlargement of the spleen and its dislocation. The gastrointestinal disturbance was first treated; the enlargement of the spleen was then treated by the administration of quinin. After the gland had decreased in size, it was replaced and fixed in its normal position by bandages and by keeping the child at rest. Intestinal peristalsis was reduced by the administration of opium. The treatment was continued for about 6 weeks. Throughout this time the patient had the abdomen elevated, and the condition was apparently overcome.

Mori[2] reports a case in which **obstruction of the intestine,** lasting for 4 days, was **caused by a wandering spleen** that was enlarged as the result of malarial infection. After repeated attempts the spleen was replaced and the intestinal obstruction disappeared. The author discusses the scanty literature relating to this condition.

E. Albert[3] reports a case of **tropical abscess of the spleen with sterile pus.** He could find but 9 cases of splenic abscess in the literature at his command. The case which he observed was seen in Algiers, in a boy of 17, who had not had dysentery. The only etiologic source of the condition seemed to be malaria, of which he had had repeated attacks, the last one having occurred 5 months previously. He was admitted to the hospital, and for a month had irregular fever, which for some weeks had been associated with severe pain in the splenic region. He was very anemic. Operation showed that pus had already been evacuated into the abdominal wall, and the cavity was easily emptied. Recovery was uninterrupted. A notable point about the case was the sterility of the pus and the fact that it chiefly consisted of small, polymorphonuclear neutrophile cells, with a few eosinophiles. Basophile elements were absent. The author considers that idiopathic abscess of the spleen is probably not so rare as the literature indicates, and believes that malaria plays an important rôle.

H. D. Rolleston[4] states that he has made a study of a number of cases of enlargement of the spleen, and finds that after having for some time maintained a recumbent posture the patients **frequently exhibit albuminuria.** This is apparently due to pressure of the enlarged spleen.

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[1] Deut. Medizinal-Zeit., Nos. 63 u. 66, 1901.

[2] Riforma med., Nos. 118 to 120, 1901.

[3] Rév. de Méd., 1901, p. 425.

[4] Lancet, Mar. 1, 1902.

DISEASES OF THE CIRCULATORY ORGANS.

METHODS OF EXAMINATION.

R. Oestreich[1] has tested the **results of percussion** of the heart in over 1000 cadavers, controlling his results by immediate autopsy. He thinks that the **relative heart dulness is of little value,** because of errors which are almost inevitable; the **absolute dulness can be determined much more exactly.** Its right border runs from the lower border of the fourth rib, at the left side of the sternum, downward and to the right, to the junction of the sternum with the fifth or sixth costal cartilage. On the left side the dulness runs from the upper point mentioned to the cardiac apex, following a somewhat convex line. There is never a really horizontal upper border. The heart dulness corresponds, as a rule, with a portion of the right ventricle, and only occasionally is it produced by a narrow strip of left ventricle. The highest point of the dulness lies a little below the situation of the pulmonary valves. The relative and absolute heart dulness correspond at the apex and at the right lower corner. Enlargement of the right ventricle causes increase in the right lower corner of the dulness, and produces a characteristic change in the right border of the heart dulness, the latter becoming wave-like or step-like. Enlargement of the right-sided dulness **should be attributed to enlargement of the right ventricle only when it shows this characteristic border-line;** some increase of the dulness toward the right may, at times, be due to increase of the left side of the heart, unless this characteristic line is observed. Enlargement toward the left may also be due to enlargement of the right heart; but if the enlargement extends beyond the mammary line, it speaks decidedly for enlargement of the left ventricle. Decrease in the size of the heart causes little change in the dulness; **enlargement of the auricles cannot be satisfactorily determined by percussion.** With pericardial effusion the highest point of the absolute dulness reaches higher and extends further from the sternum; the right and left borders of the absolute dulness then form a very markedly acute angle, as the result of pressure of the heart against the chest-wall.

G. Grote[2] discusses the methods of determining the borders of the heart. He first refers to the **phonendoscope,** and somewhat caustically criticizes the figures that have been furnished by some workers with this instrument. He believes that its most enthusiastic users themselves show its entire lack of value. He then refers to his own work with the instrument devised by Reichmann, and presents a photograph to demonstrate the following points: (1) That the **configuration and density of the internal organs have no influence upon the transmission of the sound;** (2) that the sound will be heard clearly as long as the vibrations are carried properly through the skin; and, consequently, that the condition of the skin, and particularly the tension of the skin and subcutaneous tissues, modifies the sound to an extreme degree.

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[1] Virchow's Archiv, Bd. CLX, Heft 3.

[2] Deut. med. Woch., Mar. 27, 1902.

Also symmetric figures are obtained by this method on the two sides of the body. As to the use of auscultatory percussion, the author states that the actual percussion-note against the chest-wall becomes much increased by the use of the stethoscope or phonendoscope, while the true resonance in the interior of the body is little, if at all, influenced. The consequence is that the percussion-sound itself is heard so intensely that the slight changes in the true resonance beneath cannot be well appreciated. At best, he says, in uncommon instances, where the peculiar percussion-sound given by an air-containing organ appears clearly in the percussion-note, as sometimes happens with the stomach or colon, the method may give some serviceable results. He refers to Oestreich's article as an instance of the fact that **percussion and palpatory percussion are satisfactory methods of work, if used skilfully.**

C. A. Ewald,[1] in reply to the article of Grote, insists that **auscultatory percussion is an accurate method** for demonstrating the composition of the right border of the heart. He considers it unnecessary for a skilled examiner to use this method, but thinks it valuable for teaching students.

Karfunkel,[2] in a discussion of the determination of the true **position and size of the heart and great vessels by means of the Röntgen rays,** states that the figures that he has obtained for the size of the heart are as follows: The greatest cross-diameter, 11 to 12 cm., or, in large men, 13 cm.; the length, 9 to 12 cm.; the smallest lateral diameter, 8 to 10 cm.; the greatest diagonal distance of the right ventricle from the middle line of the sternum, about 2.5 cm.—at most, 4.5 cm. Pathologic cases in his tables show marked variations from these figures. He believes that by means of the x-rays one may determine with practically absolute exactness the right and left lateral portions of the heart, and may thereby overcome many diagnostic difficulties. He also believes that he has shown that percussion gives boundaries which are of decided value only when compared with those given by the x-rays, and when established by repeated observations.

Robert Grünbaum,[3] after referring to the difficulty of counting the pulse accurately in scientific investigations, and to the difficulty that is sometimes experienced clinically in making accurate records within a given period, **recommends the use of a stop-watch,** in order that the period of observation may be kept constant and well marked.

GENERAL CONSIDERATIONS CONCERNING DISEASES OF THE HEART.

L. Braun[4] reports a case of pericarditis which presented some points that, he thinks, indicate that the **old teaching that there are six elements in the heart-sounds is incorrect;** and that, instead of the four systolic elements being due to the closure of the mitral and tricuspid valves and to the tension of the pulmonary artery and aorta, the **first sound really has only the first two elements.** The chief points in this case were that there were occasional extradiastoles evident in the pulse; but, as a rule, at the period when the extradiastoles were likely to occur, there was complete intermission of the pulse, with, however, audible and palpable contraction of the heart. With these contractions a distinct sound could be heard both at the apex and at the base, and there was no sound over the pulmonary artery or aorta. This was a direct indication that the first sound at the base comes from the heart itself, and not from the tension of the pulmonary artery and aorta.

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[1] Deut. med. Woch., May 15, 1902.

[2] Zeitschr. f. klin. Med., Hefte 3 u. 4, Bd. XLIII.

[3] Centralbl. f. innere Med., Nov. 23, 1901.

[4] Centralbl. f. innere Med., Nov. 2, 1901.

W. Ercklentz[1] gives a general discussion of **traumatic diseases of the heart,** first referring to dilation following injuries and following cardiac trauma resulting from obstruction; next to lesions of the heart-muscle due to blows upon the chest, noting cases of rupture and cases of slight injury of the heart-muscle which result in local myocarditis that ultimately leads to serious results; then to injuries of the pelvis, himself reporting the case of a man of 24, who had previously been well and engaged in heavy labor, and who, after a strain in lifting a heavy weight, exhibited rapid signs of cardiac failure and showed evidences of mitral and aortic insufficiency. From the history it was believed that the valvular disease was due to the obstruction. The author believes also that **acute and chronic endocarditis can be set up by local cardiac injury,** and describes 3 cases of chronic endocarditis which he thinks were due to such a cause, the patient in each instance having been well before the injury, and having after it exhibited signs of cardiac insufficiency and developed distinct evidences of chronic endocarditis. He then refers to ruptures of the vessels and to aneurysms following injury, and also reports a case of pericarditis following a fall upon the chest. Upon postmortem the pericarditis proved to be of tubercular origin. There was old tuberculosis of the apex of the left lung, but this was entirely healed. It was believed that the tuberculosis of the pericardium in this case was set in motion by the injury. Ercklentz discusses the literature freely, and strongly insists upon the importance of trauma in the causation of cardiac disease.

Drasche[2] reports a **number of interesting heart-murmurs.** One was a murmur of sonorous quality, resembling the sound given by the strings of a bass-viol. It was heard only over the apex. It became more marked with more energetic heart-action, and was not increased by pressure or by change in the position of the patient. The second pulmonary was not accentuated. There was a distinct thrill over the region where the murmur was heard. The patient was a man of 80, who was in a condition of extreme marasmus. Postmortem examination showed **fibrous bands running between the two layers of the pericardium,** solely at the apical region of the heart; otherwise that organ showed no changes. Apparently the murmurs were produced by vibrations of these strands, which were in such a position that with each systole they would suddenly become tense. The author believes that this case demonstrates the importance that the vibration of the valves, the chorda tendineæ, and even the arterial walls may have in the production of murmur. He then reports **chronoscopic investigations of the heart-murmur in a case of aortic regurgitation.** The diastolic murmur was found to have a duration of 0.54 second; the systolic tone had a duration of 0.36 second. These two figures do not represent the duration of either systole or diastole, but merely the duration of the sound. Drasche also reports a case of apparent **traumatic insufficiency,** in which postmortem examination showed a rupture of an aortic valve-leaflet, a part of this leaflet floating free in the blood-stream. In another case of aortic insufficiency with musical murmur necroscopy showed no explanation for the quality of the murmur; and he is inclined to believe that the murmur was due merely to abnormal currents in the blood in the enormously dilated left ventricle. He mentions a third case of aortic insufficiency with musical murmur observed directly after the strain which had apparently produced it. The man had previously been perfectly well, and after lifting a very heavy weight had pain in the precordia, sensitiveness in that region, and all the signs of severe aortic regurgitation. The musical character of the murmur disappeared while the patient was under observation. The author believes that at first a torn valve was floating in the blood-stream, and that it afterward became shrunken; hence the disappearance of the murmur.

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[1] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 5 u. 6.
[2] Wien. med. Woch., Nov. 9, 1901.

W. Gordon[1] reports a study of the **effects of posture upon heart-murmurs,** and concludes that recumbency increases hemic murmurs, with the exception of the venous hum, which it obliterates. It increases the murmurs of mitral and tricuspid regurgitation and of aortic stenosis. It decreases the murmur of mitral stenosis and scarcely affects that of aortic regurgitation. The effects of gravity and of change in the depth of the chest seem to account for the influence of recumbency. Posture should be noted in studying cases with heart-murmurs.

A. Stengel,[2] in discussing **cardiac accidents from the administration of anesthetics,** concludes that it is always difficult to tell whether the result is due to the anesthetic or to the operation. It must be recognized that basal pneumonia, gastroduodenal disturbances, and especially embolism are frequently the result of a weakened heart, and that they may, therefore, be due to anesthetics or to the shock of the operation. The evil effects of anesthetics are probably due to disturbance of the nervous or muscular mechanism, rather than to organic changes produced by the anesthetic.

T. Rumpf[3] discusses some **disturbances of the heart-function that are not due to organic disease.** He particularly emphasizes the importance of abnormal movability of the heart, which is likely to occur in patients who have been fat and subsequently have lost a good deal of weight. The result of this is a dropping of the abdominal organs and an **increased mobility of the heart.** The symptoms are usually oppression, dyspnea, and giddiness. The signs of cardiac insufficiency do not appear. The symptoms are also likely to be more marked when in the lateral position than when lying on the back. They disappear if the abdominal fat again increases in quantity. An analogous disturbance of the heart may be caused by a marked distention of the stomach with gas and similar conditions. There may be marked reflex disturbance of the heart from gall-stone disease, painful peristalsis, sexual disturbance, and the like. Some patients may even have cardiac disturbance as the result of eating special articles of food. Whatever the cause of the cardiac disturbance, the **symptoms are likely to appear with increasing frequency and readiness.** In some instances the cardiac symptoms are due purely to neurasthenia, which is especially felt by an irritable heart. Unhygienic methods of living and excess in the use of alcohol, coffee, tea, or tobacco may also be important. In some cases the cardiac disturbance is secondary to organic disease, such as contracted kidney, the gouty diathesis, etc. The diagnosis may be very questionable. The disturbance may also result from excess in sports of various kinds, such as sea-bathing, etc.

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[1] Brit. Med. Jour., Mar. 15, 1902.

[2] Am. Jour. Med. Sci., Aug., 1901.

[3] Deut. med. Woch., 1901, No. 31.

Graham Steele,[1] in a review of the **changes that have taken place during the past 30 years** in the conception of cardiac disease and its treatment, makes a number of remarks upon various features of this subject. In the first place, he insists that in aortic regurgitation direct injury to the heart occurs only during diastole, and that the **first change in aortic insufficiency is dilation.** In aortic stenosis the difficulty occurs in systole, and the author believes that as long as the ventricle is competent, dilation will not occur. He speaks of a case of **sudden death in a syphilitic child, in which coronary obstruction was found.** It is not noted whether anginoid symptoms were present in the case. He considers that the valve-lesion that is of itself of the most importance in cardiac disease is aortic insufficiency; that of next importance is mitral stenosis. He insists that the so-called Flint murmur is not a presystolic murmur, and that careful observation will show that it is a reduplicated first sound of the heart. He also states that the pulsating area close to the sternum in the second left intercostal space, which is often stated to be the left auricle, is certainly the infundibulum of the right ventricle in motion. A double diastolic sound, he insists, is positively **not due to asynchronous action of the aortic and pulmonary valves,** as demonstrated by the fact that it may be heard in very marked cases of aortic incompetence. In the determination of the presence of muscle-failure he advises dependence upon symptoms, rather than upon physical signs, as the latter are often slight and difficult or impossible to demonstrate. Cardiac percussion, while useful, is often, he believes, of little value, and it frequently deceives. Percussion should not be relied upon very greatly. He firmly states that a diastolic murmur can be produced in hearts that are simply dilated, having perfectly normal valves and orifices. In the treatment he insists upon the importance of rest, followed by properly regulated exercise, and warmly advocates the use of a proper diet. He believes that a

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[1] Quarterly Med. Jour., Aug., 1901.

largely carbohydrate diet is used altogether too much, and states that carbohydrates promote catarrhal inflammation and that a lack of proteids interferes with the proper nourishment of the heart.

A. Foxwell,[1] in a discussion of the prognosis and treatment of hypertrophy and dilation of the heart, refers to the fact that if a rheumatic valvulitis arises late in life, the prognosis is poor as compared with the cases in which the disease appears early. **Signs of venous stagnation are more serious in aortic disease than in other valvular lesions.** Enlargement of the heart due to lesions of the pericardium is of less hopeful outlook than is that due to endocarditis. The author refers to the work which he did with Billington on the effects of increasing hydraulic pressure upon the cardiac wall when there was injury of the pericardium, and to the fact that they **could produce an aneurysm in the cardiac wall merely by making a window in the pericardium over that area.** If the pericardial effusion has exceeded the ordinary capacity of the healthy pericardium, it overstretches this membrane, reduces its supporting power, and thus greatly reduces the cardiac resistance. The occurrence of dilation or hypertrophy as a result of anything but endocarditis or pericarditis due to acute infectious disease, such as rheumatism, is less hopeful than when there is endocardial or pericardial disease, for the reason that the first mentioned conditions are commonly due to myocardial degeneration. In the treatment the author insists upon the importance of remembering that heart-sufferers are exceedingly likely to be introspective, and of seeing to it that, so far as possible, the patient is not allowed to become depressed, as this has an enormous influence upon the prognosis. Massage to the precordia is very efficient in removing local pain and tenderness, and he uses general massage regularly in cases that show serious cardiac insufficiency. The author thinks that the resisted movements, as modified by the Schott brothers, have a distinct therapeutic place, but a small one. They merely bridge over the transition-stage between lying in bed and being up and about. He insists that it is usually more important to lessen the heart's work by lowering pressure than to stimulate the heart by renewed exertion.

T. C. Allbutt,[2] in a discussion of hypertrophy and dilation of the heart, refers to the case of a lad of impetuous temper, who repeatedly had attacks of half-consciousness with lividity, after marked physical overstrain, the attacks passing off very quickly and being accompanied by no evidence of organic disease of the heart. He attributes the attacks to sudden augmentation of the venous return to the heart, imperfect output, and cerebral anemia. He considers this sudden tumbling over not uncommon in the stress of school-runs, paper-chases, etc., and insists that **immature boys should not be allowed to indulge in such sports.** He then refers to some results obtained by Kerr, in an examination of school-children after ordinary running about the playground, up flights of steps, etc. Often the pulse was uncountable at the wrist for a few seconds, and it often became irregular in rate and volume. These changes are extremely frequent and probably have no evil meaning, but training greatly improves such children. The author states that for a long period he has been making careful **observations concerning the relation between blood-pressure and active exercise.** He has been more and more inclined to attribute the symptoms that occur in active exercise to tobacco, tea, alcohol, improper food, or other conditions, than to the increase of blood-pressure. The apparent heart-strain is, he believes, **not ordinarily the result of increase of blood-pressure.** It is extremely difficult to study blood-pressure during exercise, as the momentary oscillations with muscular contractions are very large. He has regularly found that most young men accustomed to active muscular exercise have habitually low arterial pressures as compared with men engaged in sedentary business occupations. The cause of the low pressure is undoubtedly the free excretion, the active metabolism, and the better nervous adjustment. Allbutt refers to the Japanese rickshaw coolies, who often run 80 or 100 miles a day while under strain, and states that Baelz, of Tokio, has told him that they apparently show no ill effects from the labor. The effect of active exercise upon the pulse is a rise, for 5 or 10 minutes, in both rate and pressure. The artery then dilates, however, and the cardiac rate falls; and this condition is usually maintained throughout the exercise. In the treatment of cardiac conditions the author refers to the Oertel and Nauheim methods, and insists that before attempting to apply any treatment we should find out the exact cause of the condition,—whether in the heart, in the arterial or the venous system, or in other organs,—and must remember that a certain strain upon the heart is incident to these methods. If the organ is already fagged out or overloaded, it is ridiculous to apply such measures at once; a preliminary course of treatment and of investigation of the conditions should precede such methods. Subsequently they may be well used in many cases. The author divides senile arterial disease into a group in which death occurs from either apoplexy or cardiac failure, and one in which there occurs an uneventful dotage. In the first, if we are fortunate enough to see the patient early and skilful enough to recognize the existence of high blood-pressure, much may be done for the patient; but it must be remembered that practically the only symptom at such a time is high blood-pressure, and that the patient may be entirely free from other complaints. Later, the author has found the effects of treatment to be decidedly unsatisfactory, even when there is only moderate arterial disease. He says that he has never seen a man fully cured whose apex had got well outside the nipple-line, although he thinks that cardiac hypertrophy may, like other hypertrophy, recede, and that the same may be true of hypertrophy of the muscular coats of the arteries.

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[1] Practitioner, Jan., 1902.

[2] Practitioner, Jan., 1902.

M. Kelsch[1] gives a description of 23 cases of **sudden death from latent cardiac and arterial disease which occurred in soldiers,** a number of the cases being of considerable interest. He insists upon the frequency of so-called idiopathic disease of the heart and of the larger vessels in soldiers, and upon the fact that they are likely to be latent in their course, to permit of the subject carrying out all the duties of a soldier, but to end in sudden death. He divides them into 3 groups: (1) Simple cardiopathies, such as hypertrophy, with or without fatty infiltration, and with or without apparent alteration of the fibers; (2) cardiopathies with valvular changes; (3) changes in the aorta, with or without concomitant alterations of the heart-valves or orifices. The causes of cardiac disease in soldiers are numerous, and are practically those that have previously been referred to by DaCosta and Fraentzel—chiefly the marked morbidity of soldiers from infections, the extreme fatigue to which they are occasionally subjected, the violent emotions of war, the evil hygiene of army life, the marked exposure, the tendency to alcoholic excesses, the poor food, and the low moral standard of soldiers.

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[1] Bull. de l'Acad. de Méd., July 30, 1901.

Ginsburg,[1] in discussing the **frequency of embolism in heart disease,** states that the reports of 250 autopsies on cases of cardiac disease show that embolism occurred in 85 cases, most commonly in the kidneys; and that both kidneys showed embolism in about two-thirds of the instances in which these organs were affected. Hematuria occurred in but 4 of these 62 renal cases, albuminuria in 26, and tube-casts in 5. Splenic embolism occurred 23 times, and produced enlargement in 9 and no symptoms in 14. Only 2 of the cases had complained of sudden, violent pain. In 15 cases the embolism was in the brain; 9 of these patients died at once, 1 lived 3 years, and 5 died in from 10 to 45 days. In 9 the heart disease was of the mitral valve; in 2, of the aortic valve; in 2 others, of both mitral and aortic; and in 2 there was myocarditis, with thrombosis in the left ventricle. There were **3 cases of embolism of the celiac.** In one of these there were hematemesis and bloody stools, but no pain; in another, violent pain and meteorism, but no vomiting or diarrhea; in the third there were no suggestive symptoms.

J. W. Brannan[2] reports a case of **unilateral right-sided venous thrombosis** associated with cardiac disease, in an alcoholic woman of 40, who had a history of rheumatism and of cardiac disease. She was in a condition of advanced cardiac incompetency, and showed general anasarca with extreme swelling and edema of the right arm and shoulder, and right side of the neck and body as far as the umbilicus. There was considerable fluid in the right pleural cavity. There was evident obstruction of the venous circulation on the right side, which was thought to be perhaps due to an enlarged mediastinal gland. Autopsy showed the right subclavian and right axillary veins and their small tributaries to be the seat of a firm, white and red thrombosis, and there was a firm, dark clot in the right brachial vein. There was no gross evidence of local traumatism or inflammation. The tricuspid, aortic, and mitral valves showed acute endocarditis. The situation of the thrombosis is of interest, since it was on the right side; while in 22 of the 24 cases collected by Welch the thrombosis affected the left side.

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[1] Deut. Arch. f. klin. Med., Bd. LXIX, Hefte 5 u. 6.

[2] Med. Rec., Feb. 22, 1902.

PERICARDITIS.

H. Eichhorst[1] reports a very remarkable case of what he calls **icing-heart (Zuckergussherz),** and which he considers an isolated instance. He says that he has repeatedly observed a similar condition of the spleen, even when the liver was not affected; but he knows of no other case involving the heart. The patient had had rheumatism some years before, and was probably an alcoholic. He exhibited marked signs of cardiac insufficiency, with extreme dyspnea and marked cyanosis. The cardiac dulness was enlarged. The sounds were pure, but somewhat obscure; the second pulmonary sound was increased. There were no other notable cardiac signs. The liver was decidedly enlarged. There was much albumin in the urine. The postmortem examination showed marked enlargement of the liver, congestion of the liver and kidneys, and general anemia; and the heart was covered with a dense, thick, marble-white tissue, which extended over practically the whole surface of the organ. The epicardium alone was involved, and there were no adhesions between the two layers of the pericardium. Macroscopically the heart-muscle was not notably affected; no microscopic report is given. The heart was very much enlarged; the epicardium was almost 3 mm. thick.

Irsai[2] demonstrated a case of left-sided **complete paralysis of the recurrens nerve caused by pericardial exudate.** The paralysis persisted after the disappearance of the exudate.

W. Türk[3] reported to the Internal Medicine Society, in Vienna, a case of **pericardial concretion with marked swelling of the liver and enormous enlargement of the spleen.** The enlargement of the liver was not an especial rarity, but the splenic tumor—which reached to below the level of the umbilicus and, toward the right, as far as the middle line, and which seemed to be the result of congestion and hyperplasia—was most notable. The patient was 18 years old. The author states that he has observed another case in a boy of 15, and considers it probable that young persons are especially liable to be the subjects of such enormous splenic enlargement in the rare cases in which it occurs.

Primrose[4] reports a case of **patency of the pericardium** in a patient who had lived to an advanced age. The pericardium showed a gap which measured vertically 3½ inches and transversely 3 inches. There was also absence of the right kidney and a septum in the urinary bladder.

Florence R. Sabin[5] reports a case of **tuberculous pericarditis** with effusion, in which **bacilli were found in the exudate. The patient recovered** after repeated tappings. The author insists that the case demonstrates that repeated tappings may be of the utmost value in causing recovery in serous effusions of the pericardium. Of the cells in the exudate, 98.5% were of the small mononuclear variety.

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[1] Deut. med. Woch., April 17, 1902.
[2] Wien. klin. Woch., No. 42, 1901, p. 1974.
[3] Centralbl. f. innere Med., Feb. 1, 1902, p. 140.
[4] Glasgow Med. Jour., Sept., 1901.
[5] Amer. Med., Mar. 8, 1902.

ACUTE ENDOCARDITIS.

H. Jackson[1] reports **59 cases of malignant endocarditis,** of which 43 were examined postmortem. Twenty-four of the latter occurred in men, 19 in women. Thirty-two occurred in patients between 20 and 45 years of age. The aortic valves were affected in 9 instances; the mitral, in 15; both in 10. The right side of the heart was involved 6 times and the endocardium of the ventricle 3 times. In 5 of the aortic cases there was old endocarditis; it was present in 7 of the mitral cases. The malignant disease had become implanted upon the old disease. **Enlargement of the spleen was present in all but 1 instance;** 13 times there were infarcts; twice, small abscesses. Miliary abscesses of the brain were found in a number of cases; acute purulent meningitis was also repeatedly found. Once there was an embolus of the artery of the Sylvian fossa. **Leukocytosis was regularly found,** the leukocytes usually numbering from 16,000 to 20,000. This is considered an important point in the diagnosis from malaria, tuberculosis, and typhoid fever. Leukocytosis, however, is usually present in acute rheumatism. Twenty-three cases were studied bacteriologically. In 2 no cultures were obtained; in 2 there were microorganisms which were not identified; in 8, Streptococcus pyogenes was found; in 5, the pneumococcus; in 3, Staphylococcus aureus; in 1, the colon bacillus; in 1 the streptococcus and staphylococcus, and in 1 the streptococcus with other organisms. **Organisms corresponding to gonococci were several times found** by microscopic examination of the lesions of the endocardium.

Lenhartz[2] makes **two groups of septic endocarditis:** those cases in which endocarditis is the most prominent thing in the clinical picture, and those in which endocarditis is only one of numerous prominent symptoms. He directs attention to the fact that the fresh endocarditis does not always arise at the site of old endocarditis if the latter is present; that is, the **mechanical factor is not an essential one.** He discusses 38 cases, in 7 of which endocarditis arose after urethral conditions, such as gonorrhea, mechanical insult to the urethra, etc. Septic endocarditis was observed 5 times after croupous pneumonia. The author carried out bacteriologic investigations in 28 cases, 19 of which were acute and 9 chronic; that is, the latter lasted more than 3 months. **In 16 cases bacteria were found during life;** in 9 they were found in cultures or smears at postmortem; and in 3 they were found in sections of the valves. They were principally staphylococci, streptococci, and pneumococci; the gonococcus was found once. It was usually comparatively easy to find the organism in the blood. Lenhartz particularly calls attention to the **frequency of Streptococcus parvus** as the cause of the disease, the organism being distinguished by its slow growth and the absence of an absorption-area around the colonies. He does not concur with Litten in the distinction of septic endocarditis from the malignant rheumatic form through the occurrence of suppurative foci in the former. In septic endocarditis suppuration may be entirely absent. He makes the division purely through the results of bacteriologic investigation. If pus-producing bacteria are found, the endocarditis is septic; if they are not, it is probably of rheumatic origin. Lenhartz then directs attention to the existence of a **chronic septic endocarditis** due to microorganisms commonly producing sepsis and having nothing to do with malignant rheumatism. He believes, however, that malignant rheumatic endocarditis, as such, does exist. The most important points in the diagnosis of septic endocarditis he considers to be heart-murmur, fever and general symptoms, enlargement of the spleen, and bacteriologic examination of the blood. He has had no satisfactory results from the use of Marmorek's serum.

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[1] Med. and Surg. Reports of the Boston City Hosp., vol. XI, p. 67.

[2] Münch. med. Woch., Nos. 28 and 29, 1901.

M. Litten,[1] in 19 **cases of malignant nonsuppurative endocarditis,** has found bacteria in 7 instances. Two of these were rheumatic cases, and these showed Streptococcus parvus; in other cases he found a large streptococcus, the etiology of these cases being unknown. He insists that the bacteriologic conditions vary so greatly in the different cases and show so few definite characteristics in any series of cases, as compared with others, that no distinction can be made upon this basis. He therefore regards Lenhartz as wrong in saying that if bacteriologic investigation is negative, the case is a malignant nonseptic one; while if bacteria are found, it must be considered septic. [Lenhartz's classification is certainly artificial, since, as Litten says, it depends purely upon the present technic in bacteriologic investigation; and the classification would at once fall if a common cause of rheumatism were definitely discovered and a satisfactory means of demonstrating the organism were provided. Litten's classification, however, on the basis of his own statistics, is hardly less artificial. These cases shade into each other in too indefinite a way to make it possible to classify them so strictly, particularly when the microorganisms found in the cases have, as Litten says, no distinctive characteristics in either of the classes of cases.] Litten has examined the statistics in 30,000 polyclinic cases, and in this number has found about 400 instances of heart disease. In 30 % to 35 % of the latter the cause was acute rheumatism; in 30 % more other causes could be discovered in the history; in the remainder no cause of the endocarditis could be found.

Bartel,[2] in discussing the etiology and histology of endocarditis, insists that **every case of verrucose endocarditis is of mycotic origin,** even the variety found in articular rheumatism. The organisms, however, soon die, and are often not discoverable, particularly in cases that have run a course at all chronic. There are, however, certain cases of verrucose endocarditis which arise in tuberculosis and in cachectic conditions, which are etiologically different from the ordinary form, which are **apparently not due to the direct action of microorganisms,** and which histologically show different characteristics. They are due to thrombotic deposits, and are possibly the result of the action of bacterial toxins.

E. Unger[3] reports the case of a boy of 18, who, after injuring himself, developed a severe acute coxitis, which was followed by severe swelling in other joints. It was suspected that the arthritis might be of gonorrheal origin, but it was at first thought that there was no secretion from the urethra. Subsequently, however, it was found that the external urethral opening ended blindly about 1 cm. from the meatus, that there was a hypospadias of the first degree, and that from this opening there was a free secretion of pus containing many gonococci. The boy developed the evidences of aortic regurgitation, and perhaps of mitral regurgitation. Cultures made from the blood showed **bacteria with the typical characteristics of gonococci.** The author insists upon the importance of using much blood in such examinations; of diluting the blood thoroughly with the culture-medium, in order to offset the bactericidal action of the blood; and of choosing a culture-medium which will allow of the rapid, unhindered growth of the bacteria. For the latter purpose he prefers a fluid medium.

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[1] Deut. med. Woch., May 22 and 29, 1902. [2] Wien. klin. Woch., 1900, No. 41.
[3] Deut. med. Woch., Dec. 19, 1901.

J. A. Scott[1] reports a case of **endocarditis developing during typhoid fever.** The signs of the lesion persisted at the last examination.

H. M. Cooper[2] reports a case of **infectious endocarditis treated with antistreptococcic serum.** Streptococci had been isolated from the blood. The serum seemed to do some good, but the case ended fatally. It was thought that if the serum had been used earlier a good result might have been obtained.

CHRONIC ENDOCARDITIS.

Aortic Regurgitation.—F. Strassmann[3] reports a case which he considered to be a well-established instance of **damage to the aortic valve as the result of trauma.** The patient was a man of 65, who had been kicked in the hand and chest by a horse. Up to this time he had been considered to be free of all cardiac disease, although no previous physical examination is reported. Two months after the injury he exhibited signs of cardiac insufficiency and died a few months later. The postmortem was undertaken when the body was already partly decomposed, but the following facts could be determined: There was complete adhesive pericarditis; the left ventricle was much dilated; there were sclerotic patches in the aorta and just above the aortic valve; there was a **rupture of the aorta** involving the intima and media, 2 cm. in length, which was evidently fairly old. Near this rupture of the aorta itself there was a **rupture of the anterior aortic valve** which began at the left point of attachment of the edge of the valve, advanced to about the middle of the valve, and then ran toward the right attachment of the valve. This was evidently an old lesion. Strassmann considers that the history of the case and the condition of the valve were sufficient to indicate that the kick of the horse had caused the damage to the valve. [A statement which does not seem to be sufficiently supported by the facts, as there is no testimony that the man did not have a cardiac lesion previous to his injury. It is quite possible that the injury was merely the cause of the onset of the signs of cardiac disturbance.]

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[1] Amer. Med., Oct. 5, 1901.

[2] Brit. Med. Jour., May 10, 1902.

[3] Zeitschr. f. klin. Med., Bd. XLII, Hefte 5 u. 6.

Depuis[1] discusses **ruptures of the aortic valves.** These are rare as compared with other heart lesions. They are more common in men, evidently as a result of occupation. They usually occur in persons that have old disease of the root of the aorta or of the valves, and are favored by rheumatic, puerperal, typhoid, or syphilitic infections, by alcoholism, and by tabes dorsalis. They commonly follow trauma, and **may be caused by anything which increases pressure** on the valves. Their situation, extent, and form vary. The rupture occurs at the weakest point, which may be determined by congenital or acquired factors. There are usually other changes in the aorta or in the heart. The result is, of course, insufficiency at the aortic valves. The myocardium may be strong enough to overcome the effects of the lesion. The ultimate results are, consequently, variable. Death may occur at once, and it may be difficult to determine whether it was caused by the trauma or the rupture. If death does not occur, the murmur of aortic regurgitation appears. If the rupture has been sudden, there is likely to be sudden, severe pain, with marked dyspnea; perhaps with syncope, faintness, or vertigo. In less severe cases the patient may feel vibration in the chest, and there may be the usual signs of aortic regurgitation, often associated with a systolic murmur, due to the vibration of the torn valve in the blood-current. The pulse is characteristic of aortic insufficiency, as are other arterial signs. The signs of insufficient filling of the arteries are likely to be pronounced in sudden cases. Death is likely to occur from dilation or asystole, and not infrequently it occurs with syncope, which results from the imperfect supply of blood to the brain.

W. S. Thayer[2] finds that a **presystolic or mid-diastolic murmur,** limited to the region of the apex, is **found in fully half the cases of aortic regurgitation.** The murmur does not differ from that heard in true mitral stenosis, except that it is of moderate intensity; but the systolic impulse is not tapping and the first sound is not sharp, while the pulse is large and quick. If these characteristics are present, the murmur is likely to be purely secondary to the aortic regurgitation, especially if there is no history of the acute infectious diseases which commonly cause endocarditis, and if there is well-marked arteriosclerosis. There are, however, often many of the other clinical features of actual organic mitral obstruction in cases of aortic insufficiency with marked presystolic murmur.

Campbell[3] reports a case of valvular cardiac disease in a healthy young man of 20. It was believed to have been produced by injury, as he had fallen from a scaffold 16 feet high and exhibited the signs of aortic regurgitation. He was known to have been examined about 4 weeks before the accident, and to have been found normal at that time. In the case there was a **suspicion of both aortic and pulmonary regurgitation.**

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[1] Gaz. Hebd. de Méd. et de Chir., 1901, No.72.

[2] Am. Jour. Med. Sci., Nov., 1901.

[3] Brit. Med. Jour., Dec. 14, 1901.

CONGENITAL VALVULAR DISEASE.

H. van Schröter[1] reports a case of **patulous ductus Botalli,** in which there was observed a **paralysis of the recurrent laryngeal nerve,** probably the first observation of this kind as yet on record. Paralysis of the recurrent laryngeal **has previously been observed in mitral disease,** an especially important report being that of Ortner, who, because of the trouble with the nerve, made a diagnosis of mitral disease and aneurysm. In the case referred to in this paper the diagnosis was extremely difficult, but it was finally decided that there was insufficiency of the mitral and tricuspid valves; stenosis of the pulmonary, dilation of the pulmonary artery and of the ductus Botalli with compression of the left recurrent nerve. At autopsy the mitral valve was found greatly stenosed, the aortic valves were practically normal, the tricuspid valve showed vegetations and was thickened; the pulmonary valves were also thickened and covered with vegetations; the ductus arteriosus was patulous and greatly dilated, and the left recurrent nerve was found compressed between the aorta and the dilated ductus arteriosus. Microscopic examination of the nerve showed degeneration of the fibers. Schröter thinks that in difficult cases paralysis of the recurrent nerve **may perhaps be of value in determining the presence of a patulous ductus arteriosus,** more particularly when mitral stenosis may be excluded.

ANGINA PECTORIS.

Rosenbach[2] discusses **myogenous pseudostenocardia.** As an etiologic cause of stenocardia the author mentions improper or insufficient breathing. He discusses a peculiar syndrome which usually occurs in robust persons, particularly men, the attacks generally following marked muscular overexercise, chills, maintaining a leaning position—such as that used in writing—for a long time, etc. The attack begins with a sense of severe oppression, but any muscular movement greatly increases it. There is spontaneous pain and tenderness over the xiphoid and its neighborhood, and along the lower borders of the ribs. The pain is such as to lead the patients to restrict their inspirations as far as possible, and, contrary to the case in the usual attack of stenocardia, the breathing is very superficial and frequent. There is often marked painful throbbing in the vessels of the neck and in the precordia. There is a peculiar and very distressing tendency to paroxysmal gaping. The **course is wholly favorable, cardiac or vascular changes usually being absent.** The diagnosis is sometimes difficult. It is important to observe that arrhythmia is always absent, and that tachycardia is rare unless there is psychic excitement. The superficial, rapid breathing and the constant attempts to overcome the tendency to sighing respiration are also rather characteristic, as is the tenderness in the region above described, and also the fact that the patients usually feel much better when lying flat on their backs than when sitting up—contrary to the case in organic asthmas. Likewise, in spite of the fact that the condition may last several weeks, the patients remain well in appearance and capable of carrying on their work. The history of alcoholic abuse, excess in tobacco, the presence of emotional or nervous disposition, and the occurrence of the condition in persons under 30 make a functional origin more probable than an organic one. The diagnosis is made much easier by certain therapeutic procedures. Massage, hot baths, and diaphoretics overcome the myogenic attacks, and the relief afforded by an induction current is extremely striking. The pains are also relieved by antirheumatic remedies.

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[1] Zeitschr. f. klin. Med., Bd. XLIII, Hefte 1 u. 2.
[2] Therap. der Gegenwart, No. 2, 1902.

G. Voss[1] describes 2 cases which occurred in persons considerably over middle age, and in both of which attacks had occurred which at first were thought to be angina pectoris. Practically all the typical symptoms were present. The fact that the patients had marked tenderness in the back over the muscles of this region, that there was no dyspnea, and also that there were no physical signs of disease of the heart, together with the fact that one of the patients, at least, had previously had rheumatism, led Voss to the opinion that both cases were pseudoangina, and that both were instances of **rheumatism of the deep muscles of the back.**

W. Pauli and R. Kauffmann[2] discuss the symptomatology of attacks of stenocardia, describing the cases of 3 persons of advanced years who had marked arterial changes and attacks of pain in the chest, with marked precordial distress and pulse-changes during the attack, a notable point about the pains being that they had the usual radiation and also **followed the course of the carotid upward,** and that there was likewise pain in the jaw, which followed the course of the internal maxillary artery and its alveolar branches. There was no paresthesia of the skin. These attacks the authors considered to be true parenchyma-pains of the vessels, in the sense in which this expression is used by Nothnagel.

ADAMS-STOKES SYNDROME.

Friedrich[3] demonstrated a case of Adams-Stokes disease **in a woman of 21.** For 6 months she had had attacks, at frequent intervals, of unconsciousness, in association with palpitation, headache, agrypnia, restlessness, and dyspnea. The apex-beat was not dislocated. From time to time the pulse was arrhythmic, and was only 28 to 40 per minute. Electric irritation of the vagus did not change the pulse-rate if a constant current was used. If an alternating current was employed, the pulse sank to between 10 and 20 to the minute. There was increase of dulness in the aortic region, some dilation of the heart, slight systolic murmur at the apex, and moderate reduction of the hemoglobin and red corpuscles. With the attacks there were **sometimes tonic and clonic convulsions.** This was thought to be the first case in the literature which had occurred in a person under 30 years of age.

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[1] Deut. med. Woch., Oct. 3, 1901.

[2] Wien. klin. Rundschau, No. 41, 1901.

[3] Wien. klin. Woch., No. 42, 1901. p. 1974.

Robert T. Edes[1] **discusses slow pulse with especial reference to Adams-Stokes disease,** reporting 4 cases in detail. He decides that interference with the augmentor nerve-fibers at a point high in their course (usually from trauma, but occasionally from disease) has shown that it is probable that the slowed pulse is the result of paresis of the augmentor nervous system, due to a lesion anywhere from the bulb down to and including the intrinsic heart-ganglia. In the case of the strict Adams-Stokes type it is probable that the lesion is situated in the auricular ganglia and that it is degenerative. There is no clear explanation of the fact that the pulse is sometimes slow and sometimes fast, but this fact is entirely analogous to the conditions observed in degenerations of other nerve-cells. The paroxysmal manifestations of Adams-Stokes disease fit in much more easily, however, with the theory of **vagus irritation** than does the permanent condition of slowed pulse. If such a theory was accepted, it would be unnecessary to have any persistent lesions to explain the condition.

CHANGES IN PULSE-RHYTHM.

K. F. Wenckebach,[2] in discussing the **pulsus alternans,** states that he believes that it is **due to a reduction in the contractile force of the heart.** Hoffmann has recently shown that when the heart-muscle is in a hypodynamic condition contraction occurs much more rapidly than when there is normal contractility. This fact Wenckebach uses to explain pulsus alternans. He considers that, so long as the rhythm is absolutely regular, the heart beats normally; but if any slight cause for arrhythmia occurs,—and this is likely to happen with every one,—the pulsus alternans will be at once produced in the following way: A stimulation to contraction comes somewhat too early; the heart is found more decidedly hypodynamic than it was before; the contraction will, therefore, be smaller and quicker; hence a longer pause will follow before the next contraction. This contraction will be stronger because the heart has recovered somewhat; hence the contraction will last longer; the following pause will, consequently, be shorter. This series of events subsequently repeats itself.

F. Lummel[3] discusses cardiac arrhythmia at length, and reaches the conclusion that arrhythmia is frequently **due to an intensification of the normal variations that occur during respiration,** the differences from the normal being only quantitative and being the result of altered nervous conditions. This is most commonly the case in the arrhythmias of convalescence, of cardiac neuroses, and probably of many cases associated with disease of the brain. The fact that the arrhythmia is directly dependent upon the phases of respiration indicates that it is nervous in origin. The author considers that this point will probably prove of the greatest importance in determining whether an arrhythmia is purely nervous or is dependent upon organic changes.

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[1] Amer. Med., May 18, 1901, and Phila. Med. Jour., Aug. 17 to Sept. 7, 1901.

[2] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 3 u. 4.

[3] Deut. Arch. f. klin. Med., Bd. LXXII, Hefte 5 u. 6.

A. Belski[1] refers to the theory of Wenckebach concerning **systoles blocked at the auriculoventricular junction,** and reports a case which he believes constitutes added testimony of the occurrence of such blocked systoles. The patient had marked arteriocardiac sclerosis with general venous stagnation, and with pronounced attacks of tachycardia and bradycardia. The most striking point in the case was that during the attacks of tachycardia there frequently occurred a forcible systole, which was followed by a short systole, and then by a long pause, during which there were weak but very distinct pulsations in the jugular vein, the latter occurring in the rhythm of the tachycardia, but unaccompanied by any contraction of the heart so far as auscultation could determine. This, he believes, is definite evidence that there did occur systoles of the auricle which produced the venous pulsations, but were blocked at the auriculoventricular junction.

Rose[2] describes a case of **paroxysmal tachycardia.** One attack lasted for 7 days; two others, for half a day. The pulse ran from 212 to 260, the respirations being undisturbed. In the more prolonged attack there was marked heart-weakness, edema, irregularity of the pulse, and an infarct of the lung. The patient distinctly felt the onset and the end of the attacks, both of which were sudden. There had been numerous attacks besides those observed. They were frequently accompanied by severe gastric disturbance, often with an eruption of herpes on the lips, and repeatedly with paresthesias of the legs. The patient had moderate arteriosclerosis and slight enlargement of the liver; otherwise the physical examination gave normal results. Rose considers the case to be **analogous to reflex epilepsy.** In the treatment drugs were entirely unsatisfactory, as were all other measures except rest and a carefully ordered life.

H. D. Everington[3] reports a case of tachycardia in which it was noted that **deep inspirations reduced the heart-beats** as markedly as from 220 per minute to 124 in the same time.

Fuhrmann[4] reports a case of **bradycardia** in which the pulse ran from 18 to 24 in a minute. There were signs of cardiac insufficiency, cardiac enlargement, and arteriosclerosis. The heart was also irregular. The pulse-rate rose to about 60 per minute after using rest, a milk diet, and valerian. The patient had been doing severe muscular work for some time before coming under observation. The condition was attributed to dilation of the heart from overstrain.

TREATMENT OF CHRONIC CARDIAC DISEASE.

Ewald[5] reports the case of a man of 62 who had had attacks of palpitation and dyspnea, and had also had evidences of slight mitral insufficiency. He repeatedly improved under the use of digitalis, but finally showed marked insufficiency that did not react to that drug. He remained for more than 3 weeks in a most serious condition, and had become extremely edematous. He was then treated by the introduction of skin trocars, the fluid being allowed to flow off. After this, digitalis had an extremely satisfactory action, the patient began to improve rapidly, the improvement continued, and he again became able to carry on his occupation. **For about 9 months during his illness he had taken morphin** without any signs of morphinism, and he readily gave up the use of the drug. The author insists that morphin is one of the most important remedies in the treatment of cardiac disease.

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[1] Zeitschr. f. klin. Med., Bd. IV, Hefte 1 u. 2.

[2] Berl. klin. Woch., Nos. 27 and 28, 1901.

[3] Lancet, May 10, 1902.

[4] Petersb. med. Woch., No. 30, 1901.

[5] Berl. klin. Woch., No. 42, 1901.

Gottlieb and Magnus[1] have used the plethysmograph in an extensive and valuable investigation of a **series of substances of the digitalis group**—digitoxin, digitalin, convallamarin, and strophanthin (Thoms). They decide that the increase of pressure after the use of these substances is due to increase in the energy of the heart-action and in the contraction of the vessels. The latter may be general or may act upon the splanchnic group. In the latter case the blood is driven to the periphery, and the vessels are influenced by the direct contractile effect of the drugs, by passive distention produced by the increased amount of blood, and by an active reflex dilation produced by the contraction of the abdominal vessels. The actual influence upon the distribution of the blood in the body is different in the two groups of drugs. Digitoxin causes the blood to be driven from the venous into the arterial system. With strophanthin, digitalin, and the other substances, the splanchnic system contracts, but at the same time the regulatory mechanism in the periphery comes into action, and the **distribution of the blood becomes changed,** in that the blood is driven from the venous into the arterial system and from the interior to the periphery. The result is to necessitate increased cardiac activity. **With strophanthin and its analogs the peripheral vessels dilate;** consequently, with these substances, the strain upon the heart is relatively less. Both groups, however, cause an increase of pressure and an increase in the rapidity of the blood-current. These results explain the increased secretion of urine, which occurs in spite of contraction of the renal vessels. The amount of digitalis also probably determines whether the increased contractile power of the heart is greater than the resistance opposed to that organ, or whether the contrary is the case.

RUPTURE OF THE HEART.

G. A. Rorie and J. Findlay[2] report 3 cases of rupture of the left ventricle, **all of them in insane subjects.** The first patient had had no notable signs of physical disease. He had been in his usual condition, and suddenly died while in bed. Autopsy showed a rupture of the left ventricle in the line of the muscle-fibers, 1 inch in length, with no evidence of local disease at the point of rupture. Microscopically there was marked fatty degeneration. The second case was a woman of 50, with alcoholic dementia. She had no notable signs of physical disease, except a quick and rather weak pulse. She had left sick for several hours before her death. She suddenly fell, and was found dead. There was a rupture in the left ventricle ¼ inch in length. This heart also showed marked fatty change. The third case was in a man of 79, who was in fair physical health. He had a quarrel with another patient, in which he expended a good deal of effort. He then walked into another room, sat down, fell over, and died in a few moments. A rupture was found on the posterior aspect of the heart, communicating with the left ventricle. The heart was fatty.

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[1] Arch. f. exper. Path. u. Pharm., Bd. XLVII, Hefte 1 u. 2.

[2] Brit. Med. Jour., Nov. 23, 1901.

D. E. Anderson[1] reports a case of **atheromatous ulceration** of the heart, with perforation and sudden death, in a man of 62. The patient had had pain in the chest for some days, and when admitted had marked dyspnea with signs of pleurisy on the left side, but no other notable physical signs. He died suddenly on the day of admission. There was a perforation the size of a crow-quill in the posterior wall of the left ventricle, in the center of an ecchymosed patch. The perforation corresponded internally with a rigid, funnel-shaped ulcer, the size of a quarter-dollar; and a similar area, which extended as deep as the epicardium, was found lower down in the left ventricle. There was no history of tuberculosis or syphilis.

DEXTROVERSION.

Paltauf[2] reports a case which was described by Bamberger in 1888. It occurred in a man then 22 years old, who had had rheumatism and, as a result, aortic insufficiency. Percussion showed the heart dulness to be on the right side, the apex-beat being palpable at the situation of the right fifth rib, somewhat outside the nipple. Autopsy showed adhesion to the lung, the pericardium being pulled to the right. There were complete pericardial adhesions. The large vessels had normal origin and course. Such right-sided positions of the heart **should be called dextroversions,** in order to distinguish them from the cases of transposition; and **dextrocardia** should clinically be applied only to cases which are of obscure origin and in which no acquired cause can be determined.

TUBERCULOSIS OF THE HEART.

A. Moser[3] discusses the literature of tuberculosis of the heart, and reports a case in which, together with widespread tuberculosis, there was found a **thrombus at the apex of the left ventricle,** and microscopic examination showed that the endocarditis was fibrous and that the thrombus was partly organized, while sections showed the presence of giant-cells and tubercle bacilli. It was apparently the result of the formation of a **subendocardial tubercle.**

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[1] Brit. Med. Jour., Nov. 23, 1901.

[2] Wien. klin. Woch., 1901, No. 42.

[3] Med. and Surg. Reports of the Boston City Hosp., vol. xi, p. 194.

LIPOMA OF THE HEART.

A. Pasini[1] reports as a collateral discovery at autopsy, in the case of a man who had died of carcinoma of the stomach, **a round tumor of the posterior part of the wall of the left ventricle,** which upon examination proved to be a lipoma. It had produced no symptoms during life. Only 2 other such cases could be found reported. Carcinoma-metastases on the heart-wall are, on the contrary, comparatively frequent. In 32 cases the author observed 1 large macroscopic metatasis and 5 microscopic areas.

DISEASES OF THE ARTERIES.

A. Qurin[2] discusses **normal intraabdominal pressure** and the **effects of intraabdominal pressure upon the arterial circulation** of the blood. He bases his statements upon the observation of a certain number of cases clinically, and upon animal experimentation. Normally intraabdominal pressure is positive. With quiet breathing it increases during inspiration and sinks during expiration; with straining breathing it sinks during inspiration and increases during expiration. The factor which causes such marked changes in the abdominal pressure, aside from the action of the diaphragm, is the **condition of the abdominal muscles.** The state of the intraabdominal pressure during the two phases of respiration is largely dependent upon the action of the abdominal muscles. The condition of the arterial pressure when there is increase in intraabdominal pressure is as follows: The increased resistance in the abdomen compresses the veins and hinders the flow from them; consequently the heart must work harder. At first the heart is able to fulfill the extra demand. The abdominal pressure, however, becomes greater and finally the heart-muscle becomes exhausted or paralyzed. The blood-pressure then falls, or may even become *nil.* In the human subject the author found that with an increase of the abdominal pressure which entirely overcame the elasticity of the abdominal muscles, the intraabdominal pressure increased during inspiration and decreased during expiration, because under such circumstance the diaphragm alone acts in increasing the pressure. If, however, the abdominal pressure sinks so far that the tension on the abdominal muscles is relieved and their elastic power can again come into play, the contrary condition appears, and we have then the picture of straining breathing, the abdominal pressure on expiration being higher than on inspiration. Qurin also refers to an observation during animal experimentation, which, however, might readily be made in human beings—viz., the presence of a **negative abdominal pressure.** This was due to a reduction in the elasticity of the abdominal muscles through chloroform. This might, however, occur in human narcosis, and is of interest to surgeons because of the danger of sucking in air through an opening in a femoral vein, for instance.

S. Kornfeld[3] reported to the Vienna Medical Society **150 determinations of the blood-pressure made coincidently with the Gärtner**

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[1] Clin. med. ital., No. 9, 1901. [2] Deut. Arch. f. klin. Med., Bd. LXXI, Heft 1
[3] Centralbl. f. innere Med., No. 12, 1902, p. 305.

instrument and the Basch sphygmomanometer. He considered that those obtained with the tonometer showed much wider variations, and that the pressure indicated by the tonometer could not be at all compared with the pressure in the carotids. Jellinek stated that in **500 soldiers he had carried out blood-pressure determinations** with the tonometer, and had had excellent results. Basch insisted that it was very important to decide upon some one instrument that can be used for clinical and experimental purposes, in order that the results of different authors may be used collectively in drawing conclusions. Wolf stated that he had found that the **results with the tonometer depend upon the size of the ring and upon various factors** connected with the individual finger, and said that he discovered that he was able voluntarily to produce extremely varied results. Nothnagel called attention to the fact that the blood-pressure in the two radials may vary as much as 10 mm. to 20 mm. of mercury. He considered the examiner's finger the best instrument for determining the blood-pressure in practice; for scientific investigation an instrument must, of course, be employed. Basch insisted that the finger gives results approaching accuracy in only about 23 % of cases. It is essential to distinguish between **arterial tension and arterial compressibility.**

K. Hirsch[1] has made some comparative studies of the sphygmomanometer of Basch and the tonometer of Gärtner. He decides that the **tonometer does not show the absolute blood-pressure,** but only the pressure produced by the vessel-wall and the soft tissues. The figures obtained with this instrument can be used for comparison only when taken in the same person from time to time, different persons giving widely varying results. It is essential that the compression-ring of the instrument should fit the finger well, otherwise the results obtained are wholly untrustworthy. Fitting a ring to the finger is, however, a tedious process, and decidedly complicates the method. **Basch's apparatus gives more satisfactory results,** is much simpler, and can be more rapidly used.

Von Basch[2] describes a **new modification of his sphygmomanometer,** that part of the instrument which is placed upon the artery being now made of rubber and of a somewhat elongated, oval form. This is placed longitudinally upon the radial artery. It is unnecessary to determine the location of this artery.

G. L. Walton and W. E. Paul[3] give a discussion of **angina cruris (intermittent claudication)** and allied conditions, including painful cramps, and discuss the condition of the arteries in these attacks, and especially emphasize the importance of examining the pedal arteries. They decide that the paroxysmal pains of angina cruris are too constantly associated with **pulseless pedal arteries** to allow of this association being a mere coincidence. Pulseless arteries may, however, occur without pains, and pains may appear with apparently normal arteries. The paroxysmal pains are probably of vascular origin, and are due to spasm, perhaps with increased blood-pressure, acting upon vessels already partially occluded. Recurring painful cramps of constant seat probably represent a modified angina cruris. The authors consider it important to examine the dorsalis pedis and posterior tibial arteries, as well as the radial and temporal arteries, in determining the condition of the vascular system and its relation to disease in the nervous system.

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[1] Deut. Arch. f. klin. Med., Bd. LXX, Hefte 3 u. 4.

[2] Wien. med. Woch., No. 44, 1901.

[3] Boston M. and S. Jour., April 3, 1902.

Zanoni and Lattes[1] discuss **Trunicek's method of treatment for arteriosclerosis.** This depends upon the fact that calcium phosphate is readily soluble in sodium chlorid solution, and also in solutions of sodium phosphate and magnesium phosphate. Sodium chlorid is found in smaller quantities in old persons than in the young. Arteriosclerosis consists largely, in advanced stages, in the deposit of calcium phosphate in the vessels. Trunicek considered that it was possible that the main chemical change in arteriosclerosis was a reduction in the amount of sodium chlorid and the other salts named; he therefore administered a **solution containing the salts mentioned** in 10 times the percentage that they were found in blood-serum, calling the solution inorganic serum. Zanoni and Lattes describe 3 cases in which symptoms of angina pectoris and dyspnea and other symptoms due to arteriosclerosis disappeared rapidly after the subcutaneous use of 1 cc. to 2 cc. of this serum every other day.

Leopold-Levi[2] has given **injections of artificial serum** in the treatment of cerebral arteriosclerosis. The injections are said to be painless, although sometimes followed by small indurations; and the author claims that they have a useful result. The paresthesias, giddiness, and disturbances of memory and of sight are said to be decidedly improved.

L. Brunton[3] states that good results may often be obtained, in chronic increase of arterial tension, by **adding nitrates to nitrites.** In cases of high arterial pressure, especially in gout, he particularly recommends the use of potassium bicarbonate, potassium nitrate, and small doses of potassium nitrite.

J. Burke[4] contributes a study of **congenital narrrowing of the aortic system.** He first discusses the history of the literature of the subject. In referring to the etiology he states that one case only (described by v. Leyden) seems to speak for a probable development of the condition during life. As a rule, everything points toward its being congenital. The author divides the cases into the following classes: (1) Those associated with the so-called blood diseases—chlorosis, pernicious anemia, hemophilia, and hemorrhagic purpura; (2) those occurring with infectious diseases, and in which a narrowing of the aorta acts either in predisposing to the infection or in favoring a fatal issue; (3) those associated with general dystrophies, such as akromegaly and gigantism; (4) those in which the clinical picture is one of heart-disease—(*a*) associated with a small heart; (*b*) with enlargement of the heart, particularly of the left ventricle; (*c*) with enlargement of the heart and rupture of the aorta;

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[1] Gaz. degli osped., No. 6, 1902. [2] Gaz. Hebd. de Méd. et de Chir., No. 80, 1901.
[3] Deut. med. Woch., April 17, 1902.
[4] Deut. Arch. f. klin. Med., Bd. LXXI, Hefte 2 u. 3.

(*d*) with enlargement of the heart, consecutive dilation, and muscular insufficiency. He reports a case of **hemophilia with narrowness of the aorta,** which was especially remarkable for the fact that the patient had no family history of a hemorrhagic tendency; and another case which he believes favors the view of Virchow, that **congenital narrowness of the aorta predisposes to infection,** particularly malignant infection, of the endocardium. The latter patient had had rheumatism. He was taken sick on January 4, 1901, ran through the course of an acute septic endocarditis, and died on March 2d, the postmortem showing involvement of the mitral, aortic, and tricuspid valves, with adherent pericarditis. A third case is reported; this belongs to the category of purely congenital narrowness of the aortic system, with consequent cardiac insufficiency and death therefrom. In conclusion, the author states that, contrary to the views of a number of other authors, he is obliged to decide that **congenital narrowing of the aortic system is a cause of disease,** since changes dependent upon this anomaly are found both anatomically and clinically. These consist in the following: (1) Heart-changes, such as left-sided hypertrophy, dilation of the left ventricle, dilation of the left auricle, venous stagnation in the lungs, right ventricular hypertrophy, and, if the condition has persisted sufficiently long, and particularly if intercurrent disease has weakened the heart-muscle, dilation of the right ventricle; (2) arterial changes. **Arteriosclerosis is frequently observed** in these subjects at a very early period, as a consequence of the persistently high blood-pressure. The clinical signs are pallor, a feeling of constriction in the chest, dyspnea, cough and expectoration, hypertrophy with dilation of the left ventricle and consequent insufficiency, increase in the size of the lungs, congestive bronchitis, increased weakness of the right ventricle, enlargement of the liver, albuminuria, and edema. The occurrence of symptoms and the nature of the first symptoms depend upon the sex, the occupation, and various other factors. In women anemia is usually the most striking sign of the disease, and appears as either chlorosis or pernicious anemia. Probably the reduction in the amount of blood is a method of compensating for the excessively high resistance which occurs in this condition. In men this method of compensation does not occur; hence the picture is one of cardiac disease. The etiology is probably either congenital narrowness or imperfect development after birth. Theoretically the condition must be considered as a predisposing cause of infectious diseases, since it makes the subject less resistant. The actual importance of it in this regard is still doubtful, as a sufficient amount of material to allow of a decision is not at hand. The condition probably predisposes to the occurrence of valvular disease.

L. Bouveret[1] describes the case of a girl who died of uremia resulting from interstitial nephritis, and especially notes that **hypertrophy of the left ventricle was absent.** The patient was chlorotic, and exhibited, like most other chlorotics, hypoplasia of the vessels. The author considers it probable that this **general cardiovascular hypoplasia** was responsible for the absence of hypertrophy of the left ventricle, and after discussing other cases in the literature decides that vascular hypoplasia may interfere with the production of hypertrophy of the left ventricle in renal sclerosis. As a rule, hypertrophy is found entirely absent. This dystrophic influence is, he believes, the result of a congenitally deficient power of development of the vascular apparatus. If a pathologic irritation, such as customarily causes hypertrophy, appears in these cases there are no reserve forces left to produce hypertrophy. These cases usually run a very unfavorable course and rapidly end in uremia. It is not improbable that in such cases the kidneys take part in the hypoplasia of the vascular apparatus.

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[1] Gaz. Hebd. de Méd. et de Chir., No. 57, 1901.

Türk[1] reports the case of a man of 44, whose vessels in practically the entire upper half of the body were almost or quite pulseless, while the abdominal aorta and femoral arteries showed strong pulsation. The whole trunk, particularly posteriorly, was covered with distinctly tortuous, strongly pulsating arteries, in which the blood passed from below upward. The author decided that the vessels of the upper extremities had been shut off by a **deforming aortitis,** and that the descending aorta and external iliac artery had produced a collateral circulation in the upper extremity. No notable functional disturbances had resulted. The patient came to postmortem examination and the diagnosis was confirmed.

Türk[2] demonstrated the case of a patient of 56 years who exhibited a **widespread ectasis of the arteries and veins of the left arm,** due, primarily, in his opinion, to a **congenital anomaly.** The condition evidently involved not only arteries and veins, but also the capillaries. As a result of the increased amount of blood, there was evidently an abnormal growth of the skeleton, except in the regions in which pressure had caused atrophy of both bones and soft tissues. The symptoms had been much increased by the occurrence of an aortic insufficiency and hypertrophy of the left ventricle, which had greatly increased the systolic pressure in the arteries. The whole left half of the body was said by the patient to pulsate much more strongly in attacks of palpitation, and objectively one could determine that there was a left-sided hemihyperesthesia. Von Schrötter stated that in a violinist who had had enormous enlargement of the vessels of the left hand a relative cure of the condition had been attempted by systematically tying off and excising a number of vessels.

AORTIC ANEURYSM.

H. Sewall,[3] in discussing **conditions other than aortic aneurysm that determine the condition of tracheal tug,** states that he has observed it in a large number of cases other than aneurysm, and at times in persons practically healthy. He thinks that a distinctly palpable tug is not infrequently associated with left-sided pleural adhesions; diminished extensibility of the lung tends to have the same effect; and a combination of these two conditions makes it more pronounced. In health the effect of the pulsation of the aorta is to stretch the lung-tissue; while if the lung-tissue is fixed, as by pleural adhesions, the trachea is pulled upon. He believes that in normal persons the descent of the heart with the inspiratory movement of the diaphragm may press the aortic arch upon the left bronchus sufficiently to transmit the aortic pulsation to the trachea, and that this may be recognizable as tracheal tug. This is more frequent in males because of the diaphragmatic type of breathing.

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[1] Wien. klin. Woch., No. 32, 1901.

[2] Centralbl. f. innere Med., April 19, 1902, p. 413.

[3] Am. Jour. Med. Sci., Aug., 1901.

Frick[1] describes a case of aneurysm in a syphilitic man of 47, in which there was a **pressure neuritis of the intercostal nerves,** with varying conditions of anesthesia and hyperesthesia of the skin, the areas affected changing their location from time to time. The pain sensation was also occasionally delayed. The author thinks that it is possible that this **variation of the sensation in its different qualities** may be somewhat characteristic of intrathoracic aneurysm.

H. D. Arnold[2] contributes a study of the **cause of death in aneurysm** of the thoracic aorta that does not rupture. He divides the cases into (1) those in which the aneurysm is not an important factor in producing the fatal result; (2) those in which death is due to disorder of the circulation, and not to direct pressure by the aneurysm; and (3) those in which death is due to the pressure of the aneurysm upon organs of vital importance.

T. R. C. Whipham[3] reports a case of aortic aneurysm which was remarkable for the fact that it was known to have **existed for 16 years** at least, during which time the patient had ordinarily been engaged in work. The aneurysm varied most strikingly in size from time to time, and repeatedly the mass, projecting from the chest-wall, seemed to be about to rupture, even years before the patient's death. Prolonged rests with medicinal and dietetic treatment caused a remarkable degree of improvement. Finally the entire external portion of the aneurysm sloughed away, but the man lived for nearly 9 days afterward. The case ultimately **terminated by external rupture**—a somewhat uncommon cause of death in these cases.

O. T. Freer[4] reports a case of **aneurysm of the aorta simulating mediastinal abscess.** Laryngoscopic examination showed stenosis of the trachea, with a normal appearance of the mucous membrane, the stenosis, therefore, apparently being due to pressure from without. There was a history of sudden expectoration of a considerable quantity of purulent sputum at the onset of the dyspnea and stridor, and there was afterward frequent and distressing cough, with profuse expectoration. These facts suggested a mediastinal abscess, but there was no fever. A tubercular abscess seemed most probable, but tubercle bacilli were absent from the sputum. Tracheal and vocal-cord paralyses were absent, in spite of the presence of a large mass of some sort in the chest. The probable cause of the very excessive expectoration in this case was, in the

[1] Wien. klin. Woch., No. 25, 1901.

[2] Am. Jour. Med. Sci., Jan., 1902.

[3] Brit. Med. Jour., Nov. 2, 1901.

[4] Chicago Med. Recorder, Dec., 1901.

author's opinion, a high degree of negative pressure below the stenosis and resulting from it. This would aspirate a large amount of blood into the vessels of the mucosa and produce a marked passive congestion.

Reinhold[1] reports 2 cases of **rupture of an aneurysm of the ascending aorta into the superior vena cava,** and also a case of compression and secondary thrombosis of the right innominate vein by a dissecting aneurysm of the ascending aorta and transverse arch. In the latter case the only striking symptom during life was edema of the right arm and of the right half of the face. He also speaks of a case of rupture of the aorta with the production of a large dissecting aneurysm, in which there were no symptoms suggesting the exact nature of the case. The **symptomatology of rupture of an aneurysm into the superior vena cava** he gives as follows: Sudden and rapidly increasing cyanosis and edema of the entire distribution of the branches of the superior cava, while the lower part of the body remains entirely free from these signs; also a loud murmur over the point of perforation, which in both the cases reported was almost continuous, but became more marked with each systole, and which reminded him of the murmur often heard on the left side in persistence of the ductus arteriosus. In the first case this murmur could be distinctly separated from two others, one systolic and one diastolic, both of the latter being due to the aneurysm itself. The existence of the **diastolic murmur** in this case is a further demonstration of the fact that such a murmur **does not always mean aortic insufficiency.** The aortic valves were shown by the autopsy to be perfectly normal. In cases of compression-thrombosis of the superior cava the signs of venous obstruction do not appear so suddenly and do not grow so marked, and the murmur at the right of the sternum is absent. Gerhardt has laid stress upon the venous pulse in the neck as a symptom of rupture of the aorta into the cava, but in neither of these cases was there such a pulse present. The author notes that the use of gelatin in aneurysm has, in his experience, been very unsatisfactory.

Cominotti[2] reports a case which occurred in a patient of 48, who had syphilis, and who exhibited signs of compression of the vena cava, and afterward of **communication between an aneurysm of the aorta and the superior vena cava.** The symptoms which the author considers of importance in the diagnosis of perforation of an aneurysm into the superior vena cava are periodic changes in the area of dulness, which are practically synchronous with changes in the intensity of the signs of venous stagnation; a very rough systolic murmur, continuing throughout diastole; an overdistention of the venous system above the location of the communication, and insufficiency of collateral circulation in the course of the disease; as a result of the latter, the appearance of varices on the surface of the thorax: positive venous pulse; and the sudden appearance of the symptoms.

Ziegenhagen[3] exhibited to the Berlin Society for Internal Medicine a specimen from a case of aneurysm of the aorta, in which there was perforation into the vena cava. The patient was a man of 43, who was admitted with a most extreme degree of cyanosis and marked edema of the upper half of the body. There were decided dyspnea and rapidity of the pulse. A large area of dulness was found beneath the clavicle, and over this there was a thrill and a systolic murmur. Hoarseness was present, but a laryngoscopic examination could not be carried out. The diagnosis made was aortic aneurysm with compression of the vena cava. It was **thought that a rupture into the cava had not occurred, because the symptoms had appeared very slowly.**

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[1] Deut. Arch. f. klin. Med., Bd. LXXI, Heft 6. [2] Wien. klin. Woch., No. 36, 1901.
[3] Centralbl. f. innere Med., Jan. 25, 1902, p. 111.

Mesenteric Aneurysm.—L. Gallavardin[1] has collected the reported cases of **aneurysm of the mesenteric arteries,** dividing them into two groups: in the first the usual causes of aneurysm, such as syphilis and trauma, were active; in the second in 9 of the total 16 cases embolism was the cause, and resulted from infectious endocarditis. The most common situation was the superior mesenteric artery where it enters the mesentery. The aneurysm is usually small. These aneurysms very frequently rupture. The symptomatology exhibits two periods. In the period of development there is usually an absence of symptoms, although there are sometimes pains or indefinite abdominal symptoms, and sometimes severe paroxysmal attacks of pain, similar to those of aneurysm of the abdominal aorta. The tumor is rarely palpable. Compression may cause various symptoms. The presence of an aneurysm is not commonly suspected. The second period is the terminal one of rupture, which is likely to occur unless the patient dies of an intercurrent disease. Death usually take place rapidly, with quickly increasing signs of peritoneal irritation, such as severe pain, nausea, vomiting, and Hippocratic facies. Death usually occurs within a few hours. Actual diagnosis of the condition is practically impossible. In only 1 of the cases reported was the possibility of aneurysm seriously considered.

Mesenteric Embolism.—E. Aufrecht[2] reports the case of a girl of 8 with uncomplicated mitral disease, who had **sudden vomiting of blood** and intense pain in the region of the liver. Blood was passed in the stools, and there was a sharply localized area of resistance below the liver. Embolus of the superior mesenteric artery was considered to be the cause. The patient recovered.

Treatment of Aneurysm.—Lancereaux and Paulesco[3] contribute another paper on the treatment of aneurysms by **gelatin injections.** The unfavorable results which have been reported by some authors they consider to be due to the use of the method in fusiform aneurysm or to following a technic which was faulty, in that either the dose or the number of injections was too small. The dose they consider should be at least 5 grams of gelatin in substance. They believe that gelatin injections cause coagulation in the aneurysmal sac, hardening of the coats of the sac, and decrease of the pain and of the expansile pulsation. The symptoms return, however, after a few days, and the injections should be repeated until from 10 to 35 have been given. Two or 3 cannot be considered to be of any value. The authors state that the method is painless. They report 2 new cases that, in their opinion, were treated with success.

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[1] Gaz. Hebd. de Méd. et de Chir., No. 82, 1901.
[2] Deut. Arch. f. klin. Med., Bd. LXXII, Hefte 5 u. 6.
[3] Gaz. des Hôp., No. 82, 1901.

A. Vaillant[1] reports a case of aneurysm of the abdominal aorta in which he believes that a **good result was obtained by subcutaneous gelatin injections.** Three injections were used in all, at intervals of a week. They were in each case followed by a severe febrile reaction, which was evidently more marked on the following day. There were also pains in the abdomen and the lower extremities. The pulsation of the aneurysm, however, became much less marked, and could be felt only over an area of 3 cm. by 2 cm., while previously it had occupied an area of 16 cm. by 9 cm. The thrill practically vanished and the murmur grew less. The general condition showed no change.

G. Klemperer[2] demonstrated to the Berlin Medical Society a specimen of a large aortic aneurysm. The patient had been treated with gelatin injections, and the aneurysmal sac showed a **very marked deposition of fibrin,** which Klemperer thought indicated the usefulness of the gelatin treatment. In the discussion Litten insisted that aneurysms not so treated often show exactly the same conditions.

In a discussion in the Medical Society of London[3] three speakers referred to their experience with the gelatin treatment of aneurysm. One mentioned a fatal issue, with symptoms of **cerebral thrombosis;** another had had unfavorable results; the third had found evidence of consolidation in all four aneurysms in which the substance had been used. One of the cases, however, exhibited **fatal tetanus** afterward, in spite of strict antiseptic precautions in administering the gelatin.

DISEASES OF THE RESPIRATORY TRACT.

GENERAL CONSIDERATIONS CONCERNING RESPIRATORY DISEASES.

A. Robin, M. Binet, and Dupasquier[4] report a study of **respiratory interchange at high altitudes,** which was undertaken in a balloon that ascended as high as 4250 meters. The effects observed were acceleration of the pulse, increase in the frequency of the respiration according to the increase in altitude, decrease in respiratory capacity, relative decrease in carbonic-acid excretion and increase in oxygen-inhalation not in direct relation to the altitude, and fall of the respiratory quotient. The main factors, besides the altitude, which influenced the result were the degree of cold, the amount of exercise carried out before ascending, and the time at which the investigation was made; there was probably also an important individual factor.

W. Müller[5] reports some experiments concerning the **bacterial content of normal and other lungs.** He carried out his work with the utmost care as to accidental infection during postmortem; before the direct examination of the lungs he put a ligature around the trachea, removed the skin of the animal before opening the chest, and adopted various other measures of a similar kind. He tested the methods recommended by Dürck and Klipstein, and found that the two yielded almost the same results, while preliminary culture upon bouillon gave numerous positive results. He decides that both Dürck and Fr. Müller have expressed themselves with too great emphasis. **Normal lungs are not absolutely free from bacteria,** but as a rule there are in normal lungs **few, if any, bacteria that are capable of active growth.** Those that are present will often not be discovered at all if solid media are used. It is probable that a considerable number of bacteria are actually carried into the lungs and are there killed. It is important, however, to use fluid as well as solid media in studying such a question.

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[1] Gaz. hebd. des sci. méd., No. 45, 1901.

[2] Centralbl. f. innere Med., April 12, 1902, p. 388.

[3] Brit. Med. Jour., Sept. 7, 1901. [4] Bull. de l'Acad. de Méd., Dec. 3, 1901.

[5] Deut. Arch. f. klin. Med., Bd. LXX, Heft 6.

Thin,[1] in discussing the **sternocostal venous festoon,** states that he does not believe that it is necessarily associated with emphysema or other disease of the lungs. He considers that it is usually found in young men who have engaged in violent overexercise at irregular intervals, and that in these subjects the veins are, in athletic terms, soft, and consequently overdistend more readily. The dilation does not occur in ordinary hard-working men as a rule. There is probably **an especial individual predisposition** to the condition, since venules on various parts of the body are likely to be enlarged at the same time.

METHODS OF EXAMINATION.

Pal,[2] in discussing the technic of **the determination of the borders of organs,** describes a method which he has carried out with a quill-pen. The latter is moved about and provides a characteristic sound. He also used another method, in which he made a constant blowing sound by water-pressure, ausculting, at the same time, with an ordinary stethoscope. The results are, he claims, good, particularly when two organs that give much the same percussion-note lie close to each other and cannot be separated by ordinary percussion, as, for instance, the heart and liver, and the various lobes of the lungs.

E. W. Whitney[3] describes a method of physical examination which he calls **mediate palpation.** He places the phonendoscope on the chest, after removing the tubes, then lays the palmar surface of the hand upon it, and thus feels the vibrations, which have been intensified by the action of the instrument.

H. Stern[4] describes a **new percussion-mallet** intended to regulate and measure the force used in percussion.

DISEASES OF THE BRONCHI.

A. Fränkel[5] reports a very remarkable case of **obliterating fibrous bronchiolitis,** which occurred in a man of 25, who **had inhaled the**

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[1] Edinb. Med. Jour., Sept., 1901.

[2] Wien. med. Woch., No. 8, 1902.

[3] Med. Rec., July 20, 1901.

[4] Med. Rec., Dec. 7, 1901.

[5] Deut. Arch. f. klin. Med., Bd. LXXIII, Kussmaul Festschrift.

vapor of nitrous acid and showed widespread bronchitis and dilation of the lungs. He improved, and after about 10 days was practically well; but about 2 weeks after the original onset he began to have severe dyspnea and cyanosis, which increased and became intense. He died on the twenty-first day. The physical signs in the latter stage had been only diffuse râles, weak breathing over the left side, and a deep percussion-note. Autopsy showed a very widespread **obliteration of the finest bronchi** by connective-tissue plugs, with a marked collection of mucus in the larger bronchi. The alveoli showed some desquamation of epithelium. The diagnosis had been made during life on the basis of the symptoms described recently, in a similar report, by W. Lange. The origin of the condition was believed to have been in necrosis of the epithelium of the mucous membrane of the bronchioles, adhesion of the secretion to these areas, and organization of the secretion. Fränkel believes that in cases of pneumonia with delayed resolution a similar process occurs in the alveoli.

J. Thenen[1] reports the case of a man of 49, who had repeatedly had hemoptysis; and who, after a slight exposure, expectorated large masses of dark clots, which, when washed, showed a marked dendritic arrangement. When the blood was washed away, they had the appearance of **typical casts of fibrinous bronchitis,** some of them being solid and some hollow. This same process was repeated several times in the acute stage; in the subacute stage the patient repeatedly expectorated masses which looked like frog-spawn. It was notable that one apex very rapidly dilated to such an extent that the subclavicular region distinctly projected.

T. D. Arland[2] contributes a clinical **study of bronchiectasis,** based upon 40 personal cases and a study of the records of others. In nearly half the instances his own cases showed, as the cause, chronic bronchitis; in 12.5 %, pleurisy; in 10 %, pneumonia; in 7.5 %, tumor; in 5 %, foreign body and aneurysm. About 75 % of the cases occurred between the ages of 10 and 40 years; only 1.6 % were below 10 years. Hemoptysis occurred in about 50 % of the cases, and was profuse in about 28 %. In the treatment the author recommends the **inhalation of creasote vapor,** using the inexpensive commercial creasote and evaporating it in a vessel over an alcohol lamp. The vapor causes marked irritation and cough, with the result that the cavity is cleared out. The inhalation is carried out for from 10 minutes to an hour, twice a week; later, daily. Arland has used intravenous formol injections without good results. The operative results are very unsatisfactory.

M. Sänger[3] states that in the symptomatic treatment of cough the **inhalation of menthol** is often of value. He has the patient place a few crystals of menthol in a spoon, warm it over a lamp for a few seconds, and inhales the vapor. When cool, he again warms it and inhales; and repeats this several times. A strong solution may also be made in alcohol; and this may be inhaled, after dropping in the palm of the hand or on a mask. The drug may also be injected, suspended in olive oil, into the larynx, if there is much mucus. The method is of more value in chronic conditions.

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[1] Wien. med. Presse, No. 39, 1901.
[2] Practitioner, April, 1902.
[3] Therap. Monatsh., July, 1901.

DISEASES OF THE LUNGS.

Emphysema.—Riegel,[1] in discussing the treatment of chronic emphysema of the lungs, states that he has used **atropin** because of the probable relation of this condition to disorder of the vagus, and particularly the relation of the attacks of asthma to irritability of the vagus. He has also found the drug useful in bradycardia and hyperchlorhydria, in both of which conditions there is probably vagus irritation.

Pneumonia.—O. Simon[2] contributes a very interesting report of investigations undertaken under the guidance of Fr. Müller, in which he demonstrates with considerable probability that **resolution in pneumonia is an autolytic process** due to the presence of a ferment or of ferments. He shows that the pneumonic lung will exhibit autodigestion; and he thinks that this is not due to the bacteria already present in the lungs, as these were almost exclusively pneumococci, which have no proteolytic action. He demonstrated the presence of albumoses in the autolytic fluid. The nitrogen of the uncoagulable substances was so large, as compared with the total nitrogen of the albumoses, as to make it practically certain either that there must be peptone present also, or that other substances, such as amidoacids, were present. He **demonstrated the presence of tyrosin** by chemical methods, and **leucin appeared microscopically** to be present likewise. The author made the interesting observation that the juice of the lung, when added to milk, causes a precipitate in the latter, **acting like a milk-curdling ferment.** Other authors have made similar observations in relation to various organs. Simon concludes that in the self-purification of the organism the following process takes place: If cell-material dies, there are substances produced, probably in the dying cells, which actually dissolve the cell-bodies, and thus lead to their being excreted from the body. As to the amidoacids, he states that he has found them everywhere that there is a regressive metamorphosis; and that their presence, therefore, means a breaking-down of tissue. In a note at the end of the article it is stated that Müller himself has confirmed and extended the work. He has found large amounts of leucin and tyrosin, free alloxuric bodies, and also lysin and histidin.

A. Prochaska[3] reports some interesting investigations concerning the **presence of microorganisms in the blood of patients with pneumonia.** He reports in all, in this connection, 40 cases of typical fibrinous pneumonia. **In all of these he found microorganisms in the blood,** and these were almost always the Fränkel pneumococcus. In two instances the microorganisms were found usually in chains. These were probably streptococcic pneumonias; in the second generation the cultures showed a more marked streptococcic arrangement. One case ended fatally; the other recovered, but left an indurative process behind. Each of these patients had a small abscess of the finger, and from this abscess streptococci and staphylococci were isolated. Possibly the infection had taken place from this source. There was no evidence of a primary source of pyemia elsewhere, in either case. Two patients showed both pneumococci and staphylococci in the blood; both died. Both showed multiple abscesses of the lungs and kidneys. Staphylococci were present in these abscesses, and this was, therefore, probably a secondary infection with pus cocci. In one fatal case of double pneumonia there were purulent meningitis and ulcerative verrucose endocarditis of the aortic valves. From the meninges and the valve-exudate pneumococci were isolated. In one case of beginning abscess of the lung pneumococci alone were obtained postmortem. In one case of metapneumonic serous pleurisy the pneumococcus alone was isolated. Empyema caused by the pneumococcus occurred in one case. A muscle-abscess from the same cause was seen in another. In one instance an acute hemorrhagic nephritis came on with the pneumonia. The author considers that the severity of the pneumonia has no direct relation to the presence of pneumococci in the blood; many of these cases were not severe, and they were not selected cases. The freedom with which the pneumococcus grows in cultures has no relation to the severity of the disease. In all, Prochaska has now reported **90 cases of pneumonia in which microorganisms were isolated from the blood during life;** only 12 of these died. He recommends, in this method of investigation, the use of large amounts of blood (10 cc.) and the employment of bouillon. The presence of pneumococci in the blood-stream explains the metastatic process in cases that run a satisfactory course; it, of course, leaves unexplained the fact that metastasis occurs in only a few cases, and its absence in most cases. The author believes that the pneumococcus regularly circulates in the blood, and thinks that his results show with added emphasis that the Fränkel pneumococcus is the cause of pneumonia. He **has never yet found the Friedländer microorganism** in the blood.

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[1] Berl. klin. Woch., No. 4, 1902.
[2] Deut. Arch. f. klin. Med., Bd. LXX, Hefte 5 u. 6.
[3] Deut. Arch. f. klin. Med., Bd. LXX, Hefte 5 u. 6.

R. I. Cole[1] discusses the results of blood-cultures in pneumonia. After trying various methods, he decided that **inoculation in 150 cc. of sterile milk** in Erlenmeyer flasks was the most satisfactory. Thirty cases were studied, 29 of them being acute lobar pneumonia, and one pneumococcus septicemia. **Pneumococci were found in only 9 of the cases, all of which ended fatally.** The author does not consider the procedure to be of diagnostic value, except in obscure cases of central pneumonia, or in general infection with pneumococcus without pulmonary lesions. He believes that when pneumococci are recovered from the blood during life the case is likely to end fatally.

Symptomatology.—J. E. Stumpf[2] discusses **2690 cases of croupous pneumonia** which occurred in Amsterdam. The total mortality was 24.49 %. Excluding children under 10 years and those treated by full baths, which gave a very high mortality, there were 1733 cases, with 25.27 % mortality. The number of men treated was more than double the number of women. The mortality from pneumonia was in men 8.2 % of the total mortality. The percentage mortality for the two sexes was about the same, the use of alcohol in the men being largely balanced by the advanced age of the women affected. The mortality was very irregular in different years, apparently not depending upon the method of treatment. The highest mortality was in January; the highest morbidity, in May and June. The high mortality of the disease in general is largely due to its frequently affecting persons with typhoid fever, diabetes, and other conditions which severely reduce the resistance of the subject. The **influence of age was most pronounced and regular.** The mortality in the aged was about 50 %; while, in the second decennium, it was about 5 %. The cases were observed between 1886 and 1898.

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[1] Johns Hopkins Hosp. Bull., June, 1902.
[2] Neder. Tijdschr. vorr Geneeskunde, 1901.

A. Hall[1] reports a case of pneumonia in a man in whom it was believed that there was **congenital transposition of the viscera.** [Since, however, it is noted that there was old disease at the upper part of the right lung, and since what was believed to be the liver dulness was very low on the left side, there must be much question as to whether it was really a transposition of the viscera or acquired dextrocardia.]

Lipman-Wulf[2] demonstrated to the Berlin Society for Internal Medicine a patient who showed widespread and very pronounced **varices of the legs and lower part of the abdomen.** He had had pneumonia in 1873, and convalescence from this disease had been complicated by thrombosis of the veins of both legs. This had been followed by the appearance of the varices in the regions described. Hence, it was evident that there had been thrombosis of the iliac veins and of the inferior vena cava. The author notes the fact that such thromboses are not very uncommon in infectious diseases, particularly in influenza. v. Leyden, in discussion, agreed that both arterial and venous thrombosis are fairly frequent in various infectious diseases. He believed that they are produced by toxic substances which cause coagulation of the blood, the source of the toxins probably being the white blood-corpuscles.

W. R. Steiner[3] discusses the occurrence of **peripheral venous thrombosis in pneumonia** and reports 3 cases. He also reviews those previously reported, and decides that the condition is very rare, as only 38 cases could be collected. It usually occurs as a sequel, rather than as a complication, and the lower extremities are always involved. [The condition can scarcely be so rare as the literature indicates, for a large proportion of the cases are probably not reported. We have seen several instances.]

Treatment.—J. C. Wilson,[4] in discussing the treatment of pneumonia, states that his **experience with antistreptococcic serum in 36 cases was decidedly unsatisfactory.** The mortality was 28 %. Hydrotherapy was also unsatisfactory. His method of treatment is to sponge regularly with water, cold sponging being used if the temperature exceeds 103° or 104° F. Ice-bags are applied early in the disease. They relieve pain, make the patient more comfortable, and slightly reduce the temperature, but have no other effect. Alcohol is given in small quantities. A calomel purge at the beginning, morphin, and Dover's powders relieve pain and make the patient comfortable. Expectorants and digitalis are not often used; aconite and veratrum viride never. Failure of the right heart, if it does not respond to strychnin and nitroglycerin, is treated by venesection. Atropin is given for pulmonary edema. Poultices and cotton jackets are not allowed. The mortality in 188 cases was 36 %, but the use of cold baths and serumtherapy tends to make the mortality greater.

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[1] Quarterly Med. Jour., Aug., 1901.

[2] Centralbl. f. innere Med., Jan. 25, 1902, p. 111.

[3] Johns Hopkins Hosp. Bull., June, 1902.

[4] Phila. Med. Jour., Nov. 2, 1901.

E. Newell[1] reports a series of 15 cases of lobar pneumonia with a mortality of 33.5 % and another series of 30 cases with 29 recoveries. He attributes the difference in mortality to the fact that he **gave no digitalis** in the second series, but used strychnin instead.

Gazza[2] considers that the **action of digitalis and digitoxin in pneumonia** and other similar infections is due not only to its effect upon the heart, but also to its producing an increase in the circulating leukocytes. His investigations of the effect of digitalis and digitoxin show that their administration produces leukocytosis.

Perreau[3] discusses the treatment of pneumonia by **ipecac,** with the object of causing depletion of the lung. A gram of this drug is added to 120 grams of mucilage of acacia, and a tablespoonful of this mixture is given every hour for about 8 hours. Both vomiting and diarrhea should be avoided. The author states that the symptoms are relieved, and expectoration is made freer and easier. If there is any weakness of the digestive tract or of the heart, the treatment may do serious harm.

L. Weber[4] reports 9 cases of pneumonia in which he used **creasote carbonate,** with, he believes, remarkably good results in all the cases. He administered about 60 grains a day.

W. H. Thomson[5] reports a series of 18 cases of pneumonia to illustrate the usefulness of creasote carbonate in this disease. In pneumonia he gives this drug in doses of 15 grains every 2 hours, night and day.

H. J. Burwash[6] recommends the use of **superheated, dry air** in the treatment of acute, localized, infective and septic diseases, including pneumonia, pleurisy, appendicitis, and osteomyelitis; and reports a number of cases to substantiate his recommendation of the treatment.

Gangrene.—J. M. Anders and J. McFarland[7] report a case of **latent diffuse gangrene** of the lung with rupture and fetid pleurisy. The patient was a man of 69 who was alcoholic. He was taken ill on October 15th with the general symptoms of pneumonia, and went through a course similar to that usually observed in senile pneumonia, with the physical signs of consolidation of the lower lobe of the right lung. Death occurred on October 21st. There had been no fetor, and the sputum had not shown anything characteristic of gangrene. The right lung, however, was adherent to the chest-wall; and there was a loculated fetid pleurisy, the lung-tissue beneath being gangrenous.

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[1] New Orleans M. and S. Jour., Nov., 1901.

[2] Riforma med., Nos. 241 and 242, 1901.

[3] Thèse de Paris, 1901.

[4] Med. Rec., Nov. 2, 1901.

[5] Med. Rec., Feb. 1, 1902.

[6] Chicago Med. Recorder, Dec., 1901.

[7] Medicine, Feb., 1902.

Foreign Body.—L. W. Atlee[1] describes the case of a man of 29, who had localized signs of pulmonary disease in the anterior portion of the left chest, with fever, some expectoration, and a rapid loss of health. The temperature soon became hectic, and the expectoration profuse and fetid. Tubercle bacilli were absent. On the fourteenth day of observation, during a violent attack of cough, a hard body was expectorated, which proved to be **a concretion** about three-fourths of an inch long and of conical shape, the largest diameter being one-fourth of an inch. It had the general appearance of necrosed bone, and gave reactions for calcium and phosphorus.

Embolus.—J. Puterman[2] reports a case in which there were signs of **embolus** of the lung after reposition of a badly set fracture of the thigh.

Echinococcus of the Lung.—Pel[3] reports a case of echinococcus of the lungs in a man of 37 who was taken suddenly ill with chill and fever after having had hemoptysis for several days. The condition was at **first considered to be croupous pneumonia** of the right middle lobe, with dry pleurisy and with left pleural effusion. Exploratory puncture showed the presence of pus in the left chest, and an opening was made without finding pus. The patient, however, became cyanotic, had severe cough, and expectorated a bloody mass, which showed a membrane. A similar membranous structure was discovered through the operative wound, the latter being coughed out through the pleural opening. The man died after a few hours. The presence of a hydatid cyst was recognized from the substance expectorated, and postmortem examination showed compression of the vena cava by an echinococcus cyst, with a chronic adhesive pleurisy, and large cysts in the upper lobe of the right lung. The neighboring pleura and lung were infiltrated with pus.

DISEASES OF THE PLEURA.

G. Viola[4] finds that the **electric conductivity of the blood-serum** in chronic serous pleurisy is, in ordinary cases, normal or subnormal. In one case, after rapid absorption of the exudate, it was increased. In chronic nephritis, with or without uremic appearances, it was nearly always increased. The author found no constant relation between the electric conductivity, the freezing-point, and the sodium-chlorid content of the blood-serum. Investigations carried out for 12 days on the same subject showed that the electric conductivity and the molecular concentration of the blood-serum remained the same throughout this time.

A. Wolff[5] discusses the **differential diagnosis of transudates from exudates,** referring to the generally accepted statement that the latter have a specific gravity above 1.018 and contain more than 4 % of albumin; while the former have a lower specific gravity and contain less albumin. Exudates are acid in reaction, transudates are alkaline. These methods of differentiation are unsatisfactory. In examining effusions he found 2 **main groups**: those in which there were practically only polynuclear leukocytes with a few erythrocytes; those in which there were lymphocytes and red blood-cells but practically no polynuclear leukocytes. In one case he found an apparent transition from the first group to the second. He found that in tuberculosis the exudates conform practically entirely to the second group, and considers this of **importance in the diagnosis of tuberculosis.** He also believes that it indicates that the effusions in tuberculosis have hitherto improperly been considered exudates, and that they are really transudates; but they at once conform to the exudate type if through any cause infection of the pleura occurs. He believes that the toxins produced in the lung in tuberculosis have some power of rendering the vessels more permeable to fluids.

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[1] Am. Jour. Med. Sci., July, 1901.

[2] Deut. med. Woch., June 12, 1902.

[3] Berl. klin. Woch., Aug. 29, 1901.

[4] Rivista veneta di scienze med., 1901, Fasc. 8.

[5] Zeit. f. klin. Med., Hefte 5 u. 6.

Dopter and Tanton[1] discuss the **cytologic study of various serous exudates,** and particularly insist upon the importance of the cytologic investigation of pleuritic exudates in the diagnosis between simple pleurisy and tuberculous pleurisy. The same method may be used to determine whether a hydrocele is of tubercular or of other origin; the authors state that in a chronic hydrocele in which there was a marked lymphocytosis, iodin injection produced a polynucleosis. The first condition was evidently chronic; the second, acute. They also consider this method of great value in the diagnosis of disease of the central nervous system, the cerebrospinal fluid in syphilis and tuberculosis usually showing a lymphocytosis, and showing the contrary condition in acute inflammatory states. Cyto-diagnosis is also useful in ascites; and the authors likewise consider it of value in the diagnosis of arthritis, particularly in determining the existence of tuberculosis.

D. Riesman[2] reports a case of **albuminous expectoration following thoracocentesis** in a patient with sarcoma of the anterior mediastinum. The fluid was gelatinous, formed a slight coagulum upon boiling, and contained 3.5 grams of albumin (Esbach's method). The quantity expectorated was 160 cc. The author considers the condition to be due to congestion by recoil, and to be ordinarily the result of a too rapid or a too great withdrawal of fluid. If complications, particularly cardiac disease or disease of the opposite lung, are present, aspiration should be performed very slowly, and only a moderate amount should be withdrawn at one time, or several small tappings may be made successively.

R. Bernard[3] reports 4 cases of **hemorrhagic pleurisy in which subcutaneous gelatin injections** were carried out after the removal of the fluid. The collection of bloody fluid in the pleura rapidly ceased after the use of the gelatin; but he attributes the good results, not to the gelatin alone, but to the good general condition of the patients, which predisposed to recovery.

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[1] Gaz. des Hôp., No. 81, 1901.

[2] Am. Jour. Med. Sci., April, 1902.

[3] Lyon méd., Oct. 13, 1901.

G. Jochmann[1] reports a peculiar case of **spontaneous nontuberculous pneumothorax** which occurred in a man of 22 and was very notable because of the onset with practically an entire absence of symptoms, the patient entering the hospital for treatment for another condition. It was found that he had at that time a marked right-sided pneumothorax, the diagnosis being confirmed by x-ray examination. Within about 6 weeks the pneumothorax had practically entirely disappeared, when it suddenly reappeared; it then gradually disappeared again, and the man ultimately showed normal chest conditions. There was absolutely no reason for considering the presence of tuberculosis. Pleural conditions were evidently absent, because the whole lung was shown by the x-rays to be collapsed; and it could only be considered to be a case of spontaneous gas-production within the pleural cavity. There was no history of any injury in the case.

E. Gassaët[2] reports some good results from the use of **lung-extract in pneumothorax** with free pleural effusion of suppurative character. He discusses 16 other cases in which good results have been claimed from the use of lung-extract.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE MOUTH.

Verger and Abadie[3] describe the case of a man who had previously been well, who had never had syphilis, and who had never taken mercurial treatment; but who had a **painful hemorrhagic gingivitis,** which ulcerated and was accompanied by pseudomembrane formation. In the pseudomembrane they found the **fusiform bacillus described by Vincent.** The patient had not the pseudomembranous angina which is common in infection with this bacillus. By the use of potassium chlorate he was rapidly cured.

DISEASES OF THE ESOPHAGUS.

M. Einhorn[4] contributes an interesting article on **idiopathic dilation of the esophagus,** reporting 10 cases not previously recorded—a large number, when it is considered that previous records contain the reports of only about 45 cases. In only 2 of the 12 cases that Einhorn has observed was there any etiologic factor discoverable; this was, in one case, trauma, and in the other, benign stricture. The condition, however, is **usually attributable to a paralysis or atony of the esophagus,** spasmodic contracture of the cardia, or absence of the reflex relaxation of the cardia during the act of swallowing. As to the symptomatology, it consists chiefly in **dysphagia,** more or less marked, and particularly notable upon hasty swallowing; a **feeling of pressure or**

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[1] Zeitschr. f. klin. Med., Bd. XLV, Hefte 1 u. 2.
[2] Gaz. hebd. des sci. méd., No. 51, 1901.
[3] Jour. de méd. de Bordeaux, 1901, No. 50.
[4] Arch. f. Verdauungs-krankheit., Bd. VII, Hefte 4 u. 5

fulness in the chest, and sometimes **suffocative attacks while eating; cough, particularly at night,** and not infrequently associated with vomiting or regurgitation of the esophageal contents; emaciation, resulting from fear of eating sufficient food, the appetite itself remaining good. Einhorn has found the swallowing sound absent in these cases, although some other writers have found it present. Passing the tube shows that, when it is introduced from 30 cm. to 35 cm., it comes into a cavity which contains neutral or slightly acid contents, that do not show the presence of HCl or gastric ferments. Pushing the tube further, it enters the stomach, and actual gastric contents are obtained. Two tubes or fluids of different colors may be used to demonstrate the presence of the dilation. The differential diagnosis is chiefly concerned with malignant tumor, diverticulum, and antrum cardiæ. Malignant tumor that has not caused stricture will not be accompanied with dilation, the swallowing sound will be present, and there will usually be signs of cachexia; diverticulum of the lower portion of the esophagus is rare, and may be considered absent if the bougie or sound always passes readily into the stomach—the use of two tubes is valuable in case there is any doubt; enlarged antrum cardiæ may be excluded if the cavity contains as much as 200 cc. The prognosis as to prolongation of life is good; but as to absolute cure, it is bad. The treatment should be chiefly voluntary attempts to cause the food to pass on from the esophagus into the stomach, by using compression of the chest, and deep breathing, and also washing the esophagus out every evening, and general care of the health.

H. Strauss[1] reports a case of **idiopathic dilation** of the esophagus, one of carcinomatous dilation with perforation into the right bronchus, and one case which was of interest because of the difficulty in determining whether the condition was dilation of the esophagus or motor insufficiency of the stomach. There are a number of interesting clinical points in connection with the cases; more particularly, the fact that the **perforation of the esophagus into the right bronchus was definitely diagnosed**—the most distinctive sign of the existence of a perforation being the test by introducing a tube into the esophagus, the patient extinguishing a candle, held before the mouth, by blowing through this tube in energetic expiration. The seat of the perforation was indicated chiefly by the x-ray examination. Strauss also describes a **number of new instruments** which he has devised for the more accurate investigation of such cases. One of them is a collapsible rubber bulb, on the end of an esophageal tube, which is introduced in collapsed form. It is then distended in the cavity until the latter is filled. The tube and bulb are then withdrawn, the air contained in the latter escaping at this juncture into an inverted cylinder filled with water. The amount of water displaced indicates the size of the cavity. He also describes a sound which has the lower end so arranged that it can be moved from side to side in searching for the opening in an esophageal dilation. He also states that he was repeatedly successful in finding the lower opening when the dilated portion was distended with air at the same time that the rubber tube was introduced. He calls especial attention to the possibility that these so-called idiopathic dilations are **due to an abnormal course of the aorta and compression of the esophagus** by that vessel. In the treatment of the condition he insists that the use of rectal alimentation is of great importance in reducing the irritation of the esophagus. He also considers the introduction of **powdered tannin** into the sac to be of value; likewise the use of effervescent powders which aid the propulsion of the contents of the sac into the stomach.

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[1] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 3 u. 4.

Begouin[1] reports a case of **total cylindric dilation** of the esophagus. A woman of 40 had had, for nearly 20 years, difficulty in swallowing, which had increased and had finally become so extreme as to have caused very great reduction in nutrition. The food seemed to stop above the stomach, and was vomited almost immediately after being swallowed. An esophageal olivary bougie passed readily into the stomach, and contents could be obtained from that organ itself. It was thought, however, that spasm of the esophagus and of the pylorus had produced the symptoms, and gastroenterostomy was carried out. The patient died soon afterward. The whole esophagus, except at the cardia and at the upper end, was found to be much dilated.

Z. Bychowski[2] states that, in order to determine the situation of any obstruction in the esophagus, he makes use of a stomach-tube which has a centimeter scale along its surface.

DISEASES OF THE STOMACH.

General Considerations Concerning Gastric Disease.—F. Penzoldt,[3] in a **general discussion of gastric digestion in man** under various influences, describes the results which have been obtained by the workers in his school. They find that the time consumed in gastric digestion in normal persons is **not much influenced by walking, bicycle riding, or traveling** in vehicles of various kinds; or by massage faradization, or the local application of cold, either by means of the ice-bag or by taking ice internally. An increase in the amount of food protracts the time of gastric digestion, but the increased time varies according to the nature of the food, and the protraction is not in direct proportion to the increased amount; for instance, **meat causes much more marked protraction of the time than does bread,** and fluids cause even less protraction than bread. Marked reduction of the superficial body-temperature protracts the period of digestion. The appearance of free HCl is delayed by energetic exercise or by cold baths, but is not greatly influenced by other similar measures. The emptying of the stomach is hastened by using local or general warm applications; also by prolonged use of gastric lavage.

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[1] Jour. de méd. de Bordeaux, No. 47, 1901.

[2] Deut med. Woch., Jan. 9, 1902.

[3] Festschrift der Universität Erlangen zur Feier des 80. Geburtstages Sr. königl. Hoheit des Prinzregenten Luitpold von Bayern.

F. A. R. Jung[1] discusses the **frequency of heredity in gastroenteric disturbances.** His conclusions are derived from a study of the gastric conditions in 597 cases, of various kinds. Of these, 166—*i. e.*, about one-third—showed hyperacidity; the average figures for the gastric acidity were from 80 to 110, in these cases. One case, however, showed the astonishing figure of 237. Gastroptosis and enteroptosis, in women, constituted 144 cases—about one-fourth of all the cases. These conditions, in men, constituted only 3 % of the cases. Atony was found in 10 %. The author describes two groups of cases in which **gastric disturbance was found in various members of the same families.** In one group there was no similarity in the analysis of the gastric contents or in the characteristics of the disease; in the second, either the analysis of the gastric contents was similar in the different cases, or the disease was of similar character. There were 40 families, in all, in which gastric disturbances were found in more than one member, the total number of individual cases in these families amounting to 98. In 14 of the families there was no similarity in the conditions in the different members; in 26 the conditions were similar. Cases of enteroptosis were found in 27 of these 40 families; and in 13 of the 40 more than one case was found in the same family. Hyperchlorhydria was found in 62.5 %; and in 37.5 % there were several cases.

T. Justesen[2] discusses the influence of various forms of food upon the secretion of HCl, and upon the osmotic pressure in the normal human stomach. He gives a **series of curves of the HCl as obtained after the administration of various forms of test-meals.** He found, in comparing the test-meal of bread with others containing increasing quantities of meat and decreasing quantities of bread, that corresponding to the increase in meat there was an increase in the total acidity to as much as double the amount seen with bread. The free acid was somewhat less markedly increased. The time taken in digestion was constantly increased. The curve of the total HCl varied but little from that of the total acidity; the curve of the total acidity ran practically parallel with that of the free HCl, rising, however, somewhat more rapidly, and always falling more rapidly and earlier than that of the free HCl. The HCl secretion after a meat meal rose very much more rapidly than it did after a meal of bread, but the curve of the total HCl reached its maximum after about the same time, in spite of the fact that in the meat meal it reached about twice the height that it did with the bread test-meal. He also contributes some **interesting observations concerning the freezing-point of the gastric contents,** with coincident determinations of the amount of chlorids, etc. One point of importance which he discovered is that it is necessary to take the freezing-point at once after removing the contents from the stomach, otherwise further digestion goes on and the freezing-point varies considerably (usually increasing, because larger amounts of food go into solution). As a result of his studies, and those which had preceded his, he makes the following statement. **Digestion in the stomach may be considered to have the following course:** The meal when taken into the stomach contains carbohydrates partly digested by saliva. As it reaches the stomach a goodly part of the soluble substances at once go into solution, hence in almost all cases the stomach-contents at once become more or less gastrohypertonic; there is at once a secretion of water or HCl (by pure osmosis), and various substances, such as peptone, sugar, and alcohol, are more or less completely absorbed from the stomach. HCl immediately unites with organic material, chiefly the albumins. Subsequently the HCl attacks the salts of the weaker organic acids, and transforms them into chlorids; then free HCl becomes manifest. The diluting secretion of the epithelial cells persists so far that it practically always **reduces the molecular concentration of the gastric contents to a point decidedly below that of the blood-serum.** During this time various portions of the gastric contents are being passed through the pylorus, the fluids chiefly leaving the stomach, while the more solid substances remain behind. Justesen believes that there is **no alkaline secretion** from the pyloric portion of the stomach. It has previously been believed that such a secretion occurred because the mineral chlorids are decidedly increased at the end of digestion, while free HCl and the total acidity are reduced. Justesen believes this is due to osmosis of sodium chlorid itself.

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[1] Arch. f. Verdauungs-krankheiten, Bd. VIII, Hefte 1 u 2; and Am. Jour. Med. Sci., June, 1902.

[2] Zeitschr. f. klin. Med., Bd. XLII, H. 5 u. 6.

F. Volhard[1] contributes a more complete article concerning **the fat-splitting ferment of the gastric juice.** He finds that this **acts chiefly upon fats which are already in emulsion,** and this is probably the reason that previous studies of the ferments have given such doubtful and unsatisfactory results. He has found that the ferment action upon fats is not due to bacteria, since the gastric juice had a fat-splitting effect even after it was passed through a bacteriologic filter; and it is also not due to pancreatic secretions, since his later work was undertaken on a dog after establishing a Pawlow-fistula. He also was able to make a glycerin extract of the gastric mucous membrane of a pig which contained fat-splitting ferments. There is **no distinct difference between the gastric steapsin and pancreatic steapsin** in their fat-splitting action. The pancreatic ferment has, however, the advantage of being able to emulsify fats, hence its effects are much more marked and constant. The gastric steapsin has some effect upon numerous neutral fats which are not emulsified, but it probably acts only upon those fats which are readily emulsified, and the more perfect the emulsion, the more marked is the action of the ferment. The ferment is excreted chiefly by the fundus. A pepsin-HCl mixture interferes with the action of the ferment, particularly when it is strong; and if it is decidedly rich in pepsin or acid, it wholly overcomes the action of the ferment. The presence of this ferment in the stomach may be considered to be an important support for Pflüger's theory that all fats must be split before they are absorbed. Previous teaching against this view has been largely based upon the fact that even when the pancreas was removed a good deal of milk fat in particular was absorbed, and this could not have been due to the bacteria in the intestines. It is, however, probable that the fat under such circumstances is digested by the fat-splitting ferment of the stomach.

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[1] Zeitschr. f. klin. Med., Bd. XLII, Hefte 5 u. 6.

F. Volhard[1] refers to his previous publication concerning the fat-splitting ferment of the gastric juice. He describes a further series of careful experiments. He concludes that the fat-splitting ferment of the gastric juice is **extremely sensitive to alkalies,** while the ferment extracted from the mucous membrane by the use of glycerin is not sensitive to weak alkalies. On the other hand, the ferment when contained in the gastric juice is **much more resistant to hydrochloric acid** than when it is extracted from the mucous membrane itself by using glycerin. He decides from this that the zymogen of the ferment is contained in the mucous membrane, while the ferment itself is present in the gastric juice. The activity of the fat-splitting resulting from the action of the ferment itself increases in proportion to the time of action, but not in regular intervals, and the degree of fat-splitting is dependent less upon the amount of fat present than upon the kind of fat. He found the ferment decreased in achylia, as were the milk-curdling ferment and the pepsin. Marked degrees of hyperacidity decrease the activity of the ferment.

Methods of Examination.—F. Schilling[2] found that **fat could be discovered** in recently killed calves in **various layers of the mucous membrane of the stomach,** partly in the cells and partly free between them; and also in the lymph-channels. He considers this to be confirmation of the view that fat which is in a readily emulsified form—for instance, milk fat—is partly absorbed in the stomach.

Memmi[3] has investigated the influence of various test-meals, and insists upon the importance of determining the **effect of different test-meals,** in order to reach a conclusion concerning any individual case of gastric disease, particularly as to the presence of free HCl. He also states that HCl may be determined to be present in some cases by means of dialysis, when ordinary reagents show little or none of the acid. He says that he has **often found sarcinæ in cases of gastric carcinoma,** when lactic acid was present and HCl absent; and sarcinæ and Oppler-Boas bacilli were not infrequently found together. The latter bacillus the author does not consider characteristic of carcinoma. **In the differential diagnosis** between gastralgia and ulcer he recommends that **doses of 1 gram of orthoform** be given in half a glass of water. If the pain is due merely to hyperchlorhydria resulting from gastritis or nervous disturbance, it is little influenced by orthoform; but in ulcer Memmi finds that the pain disappears for 3 or 4 hours. He considers that by determining the position of the patient in which orthoform works most successfully, one may decide upon the location of the ulcer.

F. Seiler[4] discusses a **new method for the investigation of the**

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[1] Zeitschr. f. klin. Med., Bd. XLIII, Hefte 5 u. 6.

[2] Fortschritte der Med., No. 23. 1901.

[3] Rif. med., Nos. 112 to 116, 1901.

[4] Deut. Arch. f. klin. Med., Bd. LXXI, Hefte 4 u. 5.

functions of the stomach, particularly for the determination of the motor power of that organ. He prepares a special test-meal, containing fat in well-divided form and in known amount. At the time of the removal of the test-meal he uses the method of Mathieu in determining the amount of gastric contents. He then determines centrifugally the amount of fat in a given portion of the stomach-contents, and calculates the total amount of fat present. He thus determines the degree of motor power. He gives formulas whereby one can determine the amount of gastric juice secreted and the acidity of the actual gastric juice itself, and reports a series of cases in which he has used this method.

E. v. Koziczkowsky[1] has investigated the value of Seiler's method for testing the function of the stomach, and finds that while the test-meal recommended seems to remain homogeneously mixed when outside the body, if prepared with great care, the investigations that he carried on concerning its behavior in the stomach show, on the contrary, that the meal apparently soon becomes more or less broken up, and, after removal and standing for a short time, is not at all homogeneous. **When it was removed at different periods, very different results were obtained** as regards the fat-content. The author is strongly inclined to believe that the method is valueless; for, did the meal not remain homogeneous in the stomach, varying portions of fat or fluid would be removed, and the portion obtained would be absolutely no indication of the motor or secretory activity of the stomach.

E. Riegler[2] reports **a new method for the estimation of the HCl** of the gastric contents. He adds barium carbonate to the gastric juice, evaporates on the water-bath, incinerates at low heat, extracts repeatedly with water, adds hydriodic acid, collects and washes the precipitate, introduces it into his urea apparatus, and adds hydrazin sulfate solution. Decomposition occurs, and nitrogen gas is given off; and from the amount of this gas the amount of HCl is calculated, a table being given for the latter purpose. [The method is necessarily inaccurate. It is difficult to carry out carefully, and would also take a considerable amount of time. The author himself states that the lactic acid may be estimated by extracting with ether in carrying out this method on the evaporated ether extract. Hence it is impossible to see how one can determine the HCl in this way and exclude the organic acids.]

L. Leney,[3] in discussing **gastric flatulence and its relation to the gastric secretion,** refers to the results which have been obtained at the University College, through a series of years past, in the **estimation of the volatile acids** of the stomach. Diluted gastric contents are placed in a flask; superheated steam is driven through the flask; and the volatile acids are collected in the flask into which the distillate goes. The volatile acidity in 23 out of 33 examinations of the normal stomach was from 0.01 % to 0.02 %, expressed in grams of solid sodium hydrate. There seems, therefore, to be a slight amount of fermentation in the normal stomach, the volatile acidity varying inversely with the amount of free HCl. In some cases in which the motility was poor, but the HCl normal, the volatile acids were found normal; in other cases they were found to be increased.

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[1] Deut. med. Woch., June 26, 1902.
[2] Deut. med. Woch., June 19, 1902.
[3] Practitioner, Oct., 1901.

J. de Groot[1] discusses **gastric fermentation,** considering carbohydrate fermentation to be the **cause of motor insufficiency, pyloric spasm, hyperchlorhydria, and ulcer.** In a number of cases of carbohydrate fermentation the author found not only alcohol and aldehyds, but also H_2S, as an evidence of the decomposition of albumins. There were likewise ammoniac compounds. The test used for the investigation of alcoholic fermentation is not the ordinary test-meal, but zwieback (30 grams with 10 grams of cane-sugar and 250 cc. of water). This was removed after an hour, and neutralized before being distilled. The therapy should consist in excluding carbohydrates; and this, in cases in which fermentation is the main element, is followed by an almost immediate relief of the symptoms. When fermentation is secondary to some other condition, improvement, but not cure, is obtained in the same way.

R. Link[2] reports some investigations concerning the **emptying of the stomach in different positions of the body.** He finds that the stomach is more readily emptied when a person lies on the right side; and that, in investigating the motility of the stomach, the position of the patient should be more carefully observed than it has been. If there is but slight difference in the amount retained when the left-sided position is assumed, compared with that retained when in the right-sided position, this may perhaps be a useful sign of pyloric stenosis. The assumption of the right lateral position after taking food may have useful therapeutic results.

Oehler[3] recommends the following **method for inflation of the stomach.** The nozzle of a Politzer bag is introduced into one nostril, and the other is tightly closed. The patient makes swallowing movements, and the bag is pressed upon at the same time. This is repeated two or three times, and the stomach is thus sufficiently inflated to be properly examined. [The method would seem to be dangerous, because of the possibility of causing serious middle-ear trouble.]

Quirolo[4] discusses the **diagnosis of insufficiency of the pylorus,** and recommends the use of his special apparatus for this purpose. He introduces the tube, inflates the rubber bulb, and then percusses over it. Ordinarily, the results of the percussion are registered at once on a drum; in pyloric insufficiency, however, the percussion wave is not transmitted to the rubber bag, because of the lack of normal compression of the air through the contraction of the stomach, the air readily escaping into the bowel.

Schüle[5] discusses the **diagnostic importance of the splashing sound** over the stomach. If it is obtained only by deep succession it is of no importance; if, however, it is obtained by a very superficial succussion, it cannot be considered normal, and it is believed by the author to indicate atony of the stomach, which is found in gastroptosis, gastric myasthenia, and gastric neurosis. It is **impossible to decide upon the degree of atony** that exists by observing the degree of the splashing sound, as the latter may be very marked in cases of very slight weakness of the gastric wall.

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[1] Nederl. Tydschrift voor Geneeskundd, II, p. 405.

[2] Deut. Arch. f. klin. Med., Bd. LXXI, Hefte 2 u. 3.

[3] Abstract, Pacific Med. Jour., Aug., 1901.

[4] Klin.-therap. Woch., No. 49, 1901.

[5] Arch. f. Verdauungs-krankheiten, Bd. VII, Hefte 4 u. 5.

Stiller[1] insists that the splashing sound is of importance in the diagnosis of gastric disease. The use of the stomach-tube only shows whether the stomach has retained the power of emptying itself; it does not indicate whether or not mild gastric atony is present. The best tests for atony are taking varying quantities of water, and determining that the lower border of the stomach descends more than is normal (Dehio); or the simpler test recommended by Stiller himself, which is the determination of a **change in the percussion-note when the patient lies upon the left side.** If the latter test is positive at the height of digestion, it indicates atony, if it is still positive after this period, it indicates motor insufficiency; if positive after fasting, it indicates dilation. Stiller considers the splashing sound to be an indication of the presence of undue distensibility or of atony, even if stagnation is not yet present. He believes that a normal stomach, even at the height of digestion, does not, as a rule, give a splashing sound; or gives one only after violent succussion.

Kuttner[2] believes that the succussion splash over the stomach is of **decided clinical importance,** and thinks that when it appears with a fasting stomach or after taking small amounts of fluid, and is elicited by superficial palpation, it is distinctly a pathologic symptom. It indicates reduction of the muscle tone. It readily occurs in gastroenteroptosis, for atony often accompanies gastroptosis or precedes it. The atony may not be evident under the ordinary circumstances of life; but after an extra strain of any kind is thrown upon the digestive tract, through acute disease or other factors, atony will appear.

A. Rosenbaum[3] believes that the splashing sound is of distinct importance in indicating the existence of gastric atony. One must know, however, exactly what the patient has taken previous to the examination. If the splashing sound is heard for a short time after taking 250 cc. or 300 cc. of fluid, and then soon disappears, it is an indication that there is gastric atony; but if the sound persists practically constantly, it indicates gastric ectasis. The author also considers that if, after taking about the amount of fluid mentioned, as much as 50 cc. can be obtained an hour later, this indicates **insufficiency of the pyloric antrum;** while the mere brief persistence of a splashing sound indicates **insufficiency of the fundus.** That the latter can occur, as distinct from insufficiency of the pylorus, is, he believes, indicated by a series of cases of carcinoma of the fundus without involvement of the pylorus, which he describes with some detail. He believes that atonic estasis can occur, and reports several cases.

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[1] Berl. klin. Woch., No. 39, 1901.

[2] Berl. klin. Woch., No. 50, 1901.

[3] Deut. med. Woch., June 19, 1902.

Schupfer[1] has investigated the **value of iodipin** in diagnosing gastric and other alimentary disease. He compared it with salol in 15 cases, and found that the results were much more trustworthy than those obtained by the use of the latter. **With disturbance in the flow of the bile, however, iodipin gives uncertain results,** and an iodin reaction never appears in the saliva in case the biliary passages are completely obstructed. Pancreatic secretion has a marked influence upon the decomposition of salol, but no effect upon iodin; hence, by using both salol and iodipin one may reach some conclusions as to the presence or absence of obstruction of the pancreatic duct. Iodipin should always be tested in a normal person, as the preparations of it vary greatly. The results obtained with it are not absolutely satisfactory, but it is a useful method when the stomach-tube cannot be safely used.

M. I. Knapp[2] states that if a patient is laid upon an ordinary examining table with the abdomen uncovered, one can, with quiet breathing, **see the contour of the stomach** by bringing his eyes about on a level with the abdomen and noticing the delicate line that moves up and down, and indicates the greater curvature of the stomach. By noting the position of this line, and percussing with two fingers,—one placed just above, and the other just below the line,—one may determine that there is a difference in note. For this reason the author considers it unessential, even in fat persons, to inflate the stomach.

F. Umber[3] discusses the **fermentative splitting of the nucleoproteids** in metabolism, a subject which has not been successfully studied, and which is difficult to get at. The chief result of his studies was the apparently satisfactory demonstration that in both peptic and tryptic digestion the nucleoproteids are split at once into nucleinic acid and pure albumin. Tryptic action was much more rapid than peptic, but apparently not more complete. The proteid product then underwent the usual course of digestion of albumins; the further course of the nucleinic acid could not be followed definitely, and it is not known through what course it advances. [The observation that pepsin has an active digestive action upon nucleoproteids is interesting, because of A. Schmidt's recent statement that a test as to whether peptic or tryptic digestion is at fault is determining whether the nuclei of cells are properly digested in their passage through the digestive tract. If undigested nuclei are found in large numbers, it indicates that tryptic digestion is at fault, for nuclei are digested by trypsin and not by pepsin. This statement is of course based upon the general teaching that pepsin does not digest nucleoproteids. Umber's work would seem to show that the statement is incorrect.]

Einhorn[4] discusses the literature relating to the **presence of mold fungi in the stomach,** and reports 4 cases in which he has found molds in the stomach. Three of them were instances of hyperchlorhydria, and the fourth was chronic gastritis with erosions. The collections of mold appeared to the naked eye like small food remnants, and were usually of a brownish color. When examined microscopically, they were found to consist of clumps of mold and spores. Einhorn cannot state what their clinical importance is, since there were no symptoms peculiar to their presence. He believes, however, that in instances in which they are found in large numbers there must be such a considerable growth of them in the stomach as to cause irritation, or perhaps inflammation. He advises **treatment with the gastric douche,** followed by spraying with an antiseptic solution, such as silver nitrate.

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[1] Rif. med., Nos. 175 and 176, 1901. [2] Deut. med. Woch., May 1, 1902.

[3] Zeitschr. f. klin. Med., Bd. XLIII, Hefte 3 u. 4.

[4] Deut. med. Woch., Sept. 12, 1901.

Gastritis.—A. G. R. Foulerton[1] reports a case of acute **membranous gastritis due to pneumococcic infection,** apparently the first case of this kind on record. The man had persistent hemorrhage from his nose and into the skin. There was also hemorrhage from the gums. He was in a much prostrated condition, and died after a rapid fall of temperature. The stomach was found to be completely lined with a rough membrane, which in parts was almost black in color. The membrane terminated abruptly at the cardiac and pyloric ends. Pneumococci and Bacillus mesentericus vulgatus were isolated from the membranes; and the latter also from the spleen. The pneumococcus isolated was of low virulence for rabbits. The author makes some remarks upon the other cases of pneumococcic infection of the mucous membranes reported. He considers that the pathogenic action of the coccus, allowing for the difference in its situation, was much the same as when it produces a lobar pneumonia. There were **fibrinous exudation, destruction of the surface structure, and a tendency to extravasation of blood.**

Vierhuff[2] reports the case of a woman of 60 who, 15 years before, had had renal calculi, and who, for a year, had had severe gastric disturbance, associated with frequent vomiting and the necessity for limiting herself to fluid diet. HCl and ferments were found to be entirely absent. She had the evidences of chronic nephritis. She soon died of inanition. Autopsy showed calculi in both kidneys, with atrophy of those organs. The **gastric mucous membrane was greatly atrophied.** The stomach contained mucus only. Microscopically, the glands were found to be in an advanced stage of atrophy. The gastric changes were thought to be secondary to the disease of the kidneys, and were **believed to be toxic,** and comparable to the ulcerations of the intestine that sometimes occur under the same circumstances. The conditions observed in carcinoma and diabetes—perhaps also in tuberculosis and pernicious anemia—as regards the HCl secretion he considers likewise to be toxic.

Acute Dilation.—C. R. Box and C. S. Wallace[3] furnish a further contribution on acute dilation of the stomach, reporting two observations. They found **dilation of the duodenum also,** a condition which they had previously stated to be common, and which they believe indicates that the theory that the condition arises in spasms of the pylorus is untenable. In one case the condition arose in a man of 29, after an accident which had produced an arthritis of the knee that finally led to amputation. In the second case the gastric dilation occurred during the course of a severe pneumonia. Experiments on the cadaver, which consisted in distending the stomach under various circumstances, led the authors to the belief that the **causal factor is pressure of he stomach upon the duodenum,** where it crosses the spine, and not compression by the mesentery and the superior mesenteric artery. The obstruction is caused by **primary paralysis of the stomach.** The differential diagnosis is difficult and complicated, but is aided by remembering the etiologic factors and by determining the limits of the succussion splash. In the treatment the authors recommend lavage, a dorsal or right lateral position, rectal alimentation, transfusion, and strychnin or atropin injections.

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[1] Lancet, April 12, 1902.
[2] Petersb. med. Woch., No. 38, 1901.
[3] Lancet, Nov. 9, 1901.

H. C. Thompson[1] discusses the symptomatology of acute dilation of the stomach, and reports 4 cases; 2 of them in women aged 24 and 40, and 2 in men aged 26 and 46. The two older patients had carcinoma, one of the pylorus and one of the pancreas; and previous to the onset of symptoms of acute dilation a **stomach-tube had been introduced** in one case and an exploratory laparotomy had been carried out in the other. In the case of the younger man the condition **followed an extraperitoneal exploration of the kidney;** and the patient was found postmortem to have slight nephritic changes. The younger man had pneumonia of the right lower lobe, and a thick yellow exudate in both pleural cavities; in the left, especially upon the diaphragmatic surface.

J. Friedenwald[2] reports 2 cases of acute dilation of the stomach due to dietetic errors, and gives a brief discussion of the literature.

Pyloric Obstruction.—I. Boas[3] discusses what he terms **stiffening of the stomach, or spasmodic contraction of the fundus** of that organ. It is observed by placing the hand over the fundus and stroking the latter, when, from time to time, it will be found to contract more or less markedly. He **describes three grades:** one of them, only partial, lasting only a few seconds, not noted by the patient, and not accompanied by the sound of the passage of fluids through the pylorus; the second, a marked contraction, palpable, visible as a ball-like elevation, and accompanied by an easily perceptible sound, and often by very slight pain; and the third, a mountain-like rising of the stomach above the abdomen, the contraction persisting for some time, being accompanied by loud gargouillement, and being more or less sharply painful to the patient. The cause of this condition is organic or functional stenosis of the pylorus, and the author **considers the symptoms important in determining that stenosis of the pylorus is present,** particularly when the question is one of diagnosis from atony of the stomach or gastric myasthenia.

A. Hammerschlag[4] describes a case of **pyloric stenosis following poisoning with hydrochloric acid.** The patient was a man of 36 who had taken two swallows of HCl. The esophagus remained intact, but gastrectasis developed 3 months later. It was observed that HCl was absent from the stomach-contents, while lactic acid was present. The isolated action upon the pyloric region was considered to be due to the rapid passage of the HCl through the esophagus and over the greater part of the mucous membrane of the stomach, the mucous membrane being protected by a thick layer of mucus. The acid was held up at the pylorus and exerted its caustic action there. The **absence of HCl** from the stomach-contents was probably due to the toxic gastritis and the resulting severe changes in the secretory glands; the presence of lactic acid to the absence of the HCl, and the stagnation of the stomach-contents.

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[1] Lancet, Oct. 26, 1901.

[2] Amer. Med., Aug. 10, 1901.

[3] Deut. med. Woch., Mar. 6, 1902.

[4] Wien. klin. Rundschau, No. 41, 1901.

Changes in Form and Position.—Büdinger[1] reports the case of a woman of 42 who had **spasmodic contraction of the pyloric antrum,** the result being a **spastic hour-glass stomach.** The condition was apparently merely an exaggeration of the normal contractions. It, however, caused severe colicky pains and prolonged, painful regurgitation after meals. Gastroenterostomy with gastropexy was carried out, with successful results. The author recommends that in some cases of hour-glass stomach resection of the pyloric portion of the stomach should be carried out.

W. Zweig[2] gives an interesting discussion of Stiller's sign—**costa fluctuans decima.** He first refers to the fact that Meinert found only a ligamentous connection of the tenth rib in 97 of 100 cadavers, and that several others who have investigated the condition anatomically have had similar results, and that some anatomic illustrations even picture the tenth rib as movable. Stiller states that enteroptosis is practically always accompanied by gastric atony. He also considers that the costa fluctuans decima is a sign either that the patient has neurasthenia at the time, or will develop it in the future, for he believes that persons of this build are strongly predisposed to neurasthenia. Zweig adds some clinical results to the anatomic results already mentioned. He **examined 100 cases** as they appeared in the clinic, without choosing them in any way. He found 49 instances of nervous dyspepsia; among these 39 had enteroptosis and 16 had atony; in 27 cases there was enteroptosis without atony, and in 4 cases there was atony without enteroptosis. The other cases were of various kinds. The figures mentioned, however, demonstrate, he thinks, that **enteroptosis is by no means necessarily combined with atony;** indeed, the contrary is usually true. Of the 100 cases examined exactly one-half showed movable tenth rib; 27 were women and 23 men. Movable tenth rib was seen in 32 of 49 cases of nervous dyspepsia. In the 39 cases of enteroptosis it was seen 26 times. It was seen in 65 % of nervous dyspepsia in general; in 67 % of enteroptosis; and it is therefore evident that it is **not a distinctive sign of enteroptosis.** It occurs so frequently in nervous gastric disturbances as to be worthy of being called a sign of this disease, but only in a very limited sense, as it is not infrequently observed in persons who are perfectly normal, and in those who have other forms of disease than enteroptosis. It certainly is not a positive sign of neurasthenia, and it appears in men and in women with about equal frequency.

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[1] Wien. klin. Woch., No. 36, 1901.

[2] Arch. f. Verdauungs-krankheit., Bd. VII, Heft 3.

B. Stiller[1] contributes a reply to the numerous criticisms that have recently appeared of his stigma of general neurasthenia,—the **floating tenth rib.** He goes into an extensive discussion of the various points which have been brought up, and, without offering any very new facts, states that he is still convinced that the sign is not only one of enteroptosis, but of general congenital asthenia.

Stiller[2] later gives a further discussion of enteroptosis and its costal stigma. He admits that enteroptosis is, at times, such a marked factor in these cases that it really controls the whole clinical picture; but he insists, once more, that the **displacement of the abdominal organs is only a part of a general neurasthenic habitus.**

Functional and Neurotic Disturbances.—A. Pick[3] discusses the various forms of **sensory neuroses** of the stomach, noting the cases of irritability to thermic, mechanical, and chemical irritants, and especially pointing out hyperesthesia to acids. He insists that **diet is the most important means of overcoming these neuroses,** and says that it is often most essential to see that the patient eats sufficient quantities. If the patient desires special food, even though it is apparently indigestible on ordinary principles, the author recommends that it should be given, as it is usually well borne under such circumstances. Hydrotherapy and alkalies are useful. Change of occupation and of surroundings is often important. Pick considers faradization and internal gastric douches also of importance.

M. Buch[4] discusses the **nature and anatomic seat of gastralgia.** He decides that it is due to an excessive irritability of the abdominal sympathetic, and that its seat is in the epigastric portion of the sympathetic, and not in the stomach itself. Therefore he thinks that the disease should be called epigastralgia, rather than gastralgia.

Von Aldor[5] discusses **continuous gastrosuccorrhea.** He has for some time investigated the question whether a normal person shows gastric juice in the stomach after fasting. He decides that this is not the case; and that if small amounts of fluid are obtained after the introduction of the stomach-tube, it is due to the irritation produced by the stomach-tube, or the fluid is not a true gastric juice. It may contain minute quantities of HCl, but pepsin is not present in recognizable amounts. Further, this condition is absolutely distinct from Reichmann's disease, in which several hundred cubic centimeters of active gastric juice may be obtained. **If gastric juice is found in the fasting stomach, the condition is pathologic.** The author has observed 5 cases of true Reichmann's disease; he considers the condition rare. The characteristic symptom is, in his opinion, the presence of large amounts of gastric juice in the fasting stomach, the contents being of very low specific gravity. The gastric contents are extremely acid, both when obtained from the fasting stomach and when obtained after a meal. The free HCl is high in amount, and there is a high pepsin content and an excessive total acidity. As a result of the latter condition, amylolysis is incomplete. The author considers the demonstration of the **entire absence of motor insufficiency essential for the diagnosis** of Reichmann's disease. Chronic gastrosuccorrhea alone, without complications, he considers to be a definite disease. Periodic gastrosuccorrhea cannot be considered to be more than a symptom. In the treatment he considers atropin to be of much value, as are the use of alkaline and saline waters and Carlsbad salts, the reduction of the carbohydrates, increased amounts of fat in the diet, and lavage as necessary.

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[1] Arch. f. Verdauungs-krankheit., Bd. VII, Hefte 4 u. 5

[2] Deut. med. Woch., May 22 and 29, 1902.

[3] Wien. klin. Woch., Nos. 35 and 36, 1901.

[4] Arch. f. Verdauungs-Krankheit., Bd. VII, Heft 6.

[5] Berl. klin. Woch., No. 39, 1901.

H. Illoway,[1] in a **study of hyperacidity,** reports his investigations of 110 cases, inclusive of carcinoma, in which the gastric contents were examined; 23 were instances of hyperacidity, and 20 of hypoacidity. Of the cases, 67 were considered to be normal. Hyperacidity was considered to be present when the symptoms indicated it and the use of acids increased the symptoms. The figures in the cases called normal varied from 46 to 90, most of the cases lying between 50 and 80. In the cases called hyperacidity, the figures varied from 52 to 120, only 3 of them showing figures above 94. The author decides that the total acidity is not a satisfactory indication of the existence or absence of hyperacidity [a conclusion with which every one must agree]. The author also investigated the free acidity by Mintz's method in 27 normal cases and 12 cases of hyperacidity, and decides that, in this way also, it is impossible to tell definitely that hyperacidity is present. He gives a general discussion of the symptomatology of the condition, and decides that the **diagnosis can be made only upon the basis of a group of symptoms,** and not upon any one symptom. In the treatment he strongly recommends the use of very large quantities of nitrogenous food, making the astonishing statement that he attempts to tire out the stomach, and overcome a condition of irritation by making it functionate excessively for a long time. He refers to the statements of Strauss and Schüler, that in hyperacidity the contents are excessive, appear unusually fluid, have a low specific gravity, and give an abnormally marked reaction for amidulin. He says that he was unable to determine that the contents had any particular appearance in hyperacidity. The specific gravity in this condition he found usually low; but he states that the specific gravity in other cases did not bear any relation to the acidity, and that he **does not think that the acidity is directly proportionate to the specific gravity.** [Schüler's report directed attention to the fact that when symptoms of hyperacidity were present without excessive acidity of the gastric contents, the specific gravity would usually be found low. This certainly does not indicate a direct relation between the acidity and the specific gravity, but rather the contrary.] Illoway finds that the reaction for amidulin is likely to be excessive in hyperacidity; but he more especially states that the chief abnormality is that the reaction lasts for 24 to 30 hours in hyperacid gastric contents, while in normal contents it disappears in 10 to 15 hours.

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[1] Arch. f. Verdauungs-krankheit., Bd. VIII, Hefte 1 u. 2.

L. Kuttner[1] first discusses the **use of the term achylia gastrica** and the propriety of extending it to those cases in which secretion is not entirely absent. He directs attention to the fact that **secretion is entirely absent only with the most extreme rarity,** and describes a series of investigations that he has made of the amount of pepsin present, using Hammerschlag's and Mett's quantitative method for pepsin. Pepsin and pepsinogen were, in rare instances, found absent; but only when HCl was completely absent. With hyperchlorhydria and hypersecretion the amount of pepsin was found both increased and decreased, under different circumstances. It is evident, then, that there is **no direct relation between the HCl and the ferments,** since similar conditions have been observed with the milk-curdling ferment; hence, **the name achylia, as based merely upon the determination of the HCl, is of very doubtful value.** It has, further, recently been shown that the stomach has a normal specific diluting secretion, and this is another ground for objection to such a nomenclature. As to the possibility of diagnosing the existence of simple achylia gastrica and distinguishing it from a lack of secretion due to organic change, Kuttner states that he thinks **such a diagnosis is at present wholly impossible.** The two condition are so very similar in all their symptomatology that any attempt to separate them is very likely to lead one into error. The **examination of fragments of gastric mucous membrane is unsatisfactory to an extreme degree,** and Kuttner has himself become more and more positively convinced that this method has little value, agreeing fully with the statements of Leuk [abstracted from the same journal, YEAR-BOOK, 1900].

G. Korn[2] reports a number of cases of **heterochylia**—those cases in which variable conditions of the gastric contents are found. He notes that these cases demonstrate the necessity for repeated examinations of the gastric contents before important conclusions are reached.

A. Sommer[3] reports 23 cases of **gynecologic disorders** in which he investigated the conditions of the gastric contents and the position of the stomach. Gastric symptoms are very common in gynecologic troubles, but there has been comparatively little study of the actual state of the stomach. In these cases subacidity was found twice; normal conditions, once; very high normal acidity, four times; and **very marked hyperchlorhydria** in the remainder of the cases. Hence, from this standpoint alone there was an explanation for the symptoms. In a large number of the cases there was also gastroptosis; movable kidney, however, was present but twice. In only two instances were the gastric symptoms relieved by the treatment of the gynecologic disorder, and in one the gynecologic treatment made the gastric symptoms worse. The relation between the alterations in the secretion and in the position of the stomach and faulty positions of the uterus cannot be stated, because the statistics are too insignificant; but **retroflexion seems to be quite frequently associated with gastroptosis.**

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[1] Zeitschr. f. klin. Med., Bd. XLV, Hefte 1 u. 2.

[2] Arch. f. Verdauungs-krankheit., Bd. VIII, Hefte 1 u. 2.

[3] Centralbl. f. innere Med., Mar. 1, 1902.

M. Einhorn,[1] in discussing **dyspeptic asthma,** states that he has observed 31 cases in which the chest-organs were entirely normal, and the asthma seemed to be due to digestive disturbances. He divides them into: (1) the acute form of dyspeptic asthma; (2) the chronic type. In the latter case the attacks may come on immediately after meals or some hours thereafter. In the former case there is often a close resemblance to angina pectoris. He reports the gastric conditions in these cases, and notes that achylia gastrica was very frequent, and that hyperchlorhydria was likewise often seen. The dependence of the asthma upon the digestive disturbance was shown by the disappearance of the asthmatic attacks with proper care of the digestive tract. The author also notes the likelihood of attacks of asthma occurring in floating liver.

Bregmann[2] discusses the literature on **fecal vomiting in hysteria,** and describes the case of a hysteric person who had persistent constipation and vomiting of blood and feces. There was a prolapsus ani which was twice surgically treated, recurring each time.

Gastric Ulcer.—W. van Yzeren[3] gives a contribution concerning the **pathogenesis of chronic gastric ulcer.** He states that the reason previous attempts to produce gastric ulcer by means of cutting the vagus have been unsuccessful is because this is such a dangerous operation when carried out in the neck, since it then involves the cardiac and pulmonary branches. He has made a series of **sections of the vagus below the diaphragm,** and of 20 rabbits so operated upon he states that he has **produced chronic gastric ulcer in half that number.** The rabbits which showed no ulcers were in 5 instances killed only one day after the operation, while the other 5 were killed 2, 5, 9, 10, and 47 days respectively after the operation. In the 10 positive instances examination was undertaken 7, 8, 15, 41, 43, 44, 59, 169, and 289 days respectively after the operation. He gives a description of the ulcers which corresponds closely with that of ulcers as found in human subjects. In some instances the **ulcers actually perforated the stomach-wall.** He gives an extensive discussion of the movements of the stomach, and the possibility of diagnosing spasm of the pyloric region of the stomach in animals and in human beings, and states that he believes that the method by which vagotomy produces gastric ulcer is through causing **spasm of the pyloric portion** of the stomach and the production of slight hemorrhages. Reasoning from this, he **advises operation in gastric ulcer** when at all resistant to treatment, and proposes a new operation which he has carried out on rabbits; *i. e.*, division of the muscular coat of the pyloric portion of the stomach, and of the pylorus without dividing the mucosa or the muscularis mucosa. He believes that this has prevented the occurrence of ulcer in a number of animals, and that it is a rational and safe operation. He thinks that **pyloric spasm can be diagnosed in human beings** oftentimes by palpating the pyloric region of the stomach and by demonstrating intermittent retention in the stomach. [The report of the experiments is not convincing. The author's reasons for accepting the existence of spasm of the pyloric region of the stomach are still less convincing, and until such spasm has been satisfactorily shown to exist it would be more than rash to operate at once on gastric ulcer, after the method described, or any other, merely because the case did not respond rapidly to treatment.]

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[1] Jour. Am. Med. Assoc., Feb. 1, 1902.
[2] Neurologisches Centralbl., No. 19, 1901.
[3] Zeitschr. f. klin. Med., Bd. XLIII, Hefte 3 u. 4.

G. Fütterer,[1] in a discussion of chlorosis, reports a series of experiments on dogs, in which he **removed portions of the mucous membrane of the stomach** without any other procedure, and also coincidently and subsequently **abstracted large quantities of blood;** he likewise, in other animals, injected pyrogallic acid. After repeatedly removing blood, an unhealed ulcer was found after 3 weeks; but the ulcer had a tendency to heal, and therefore was not a characteristic gastric ulcer. After injections of pyrogallic acid in a dog and a rabbit, typical gastric ulcers were found, which had no tendency to heal. In the treatment of chlorosis the author especially recommends the use of beef-juice. In inoculating animals with suspected matter for the purpose of diagnosing tuberculosis, he recommends the previous production of artificial anemia.

Wiesinger[2] reports a **remarkable case of perforation of a gastric ulcer** in a man of 36, the patient coming into the hospital after 4 days had passed. He was operated upon at once. The peritoneal cavity was found to be filled with foul pus and great masses of fibrin. After cleansing as thoroughly as possible, a large perforation was found on the lesser curvature, and was inverted into the stomach-cavity. A Mikulicz tampon was then introduced, the wound being otherwise closed. The patient did well for some days, but the wound became infected from the abdominal cavity and had to be opened. A few days later there was a **prolapse of numerous folds of intestine.** The prolapse was replaced under narcosis. Three days later there were signs of **intestinal obstruction;** the intestines were punctured through the wound to draw off the gas. After collapse of the intestine pus escaped from the upper end of the wound, and after this a large **intraabdominal abscess was opened. Signs of subphrenic abscess** developed a few days later. Puncture was negative, but shortly afterward the patient expectorated about half a pint of foul pus. Subsequently puncture was positive. The ninth rib was resected, and an abscess-cavity containing a pint of pus was opened. The patient then proceeded to complete recovery. The author also notes a case which he had seen some time before, in which **perforation of a gastric ulcer occurred and the patient evidently had had peritonitis for 6 weeks** at the time when he first saw her. When seen at that time, the abdomen was tympanitic, except in the region of the right anterior superior spine, at which point the edge of the liver could be felt. The normal liver dulness was entirely gone; there was a loud, tympanitic percussion note up to the fourth rib. Operation was undertaken, and a cavity containing 3 or 4 liters of fluid and much gas was opened. The cavity was tamponed, and **the woman recovered entirely.**

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[1] Chicago Med. Recorder, July, 1901.

[2] Deut. med. Woch., Jan. 30, 1902.

A. Hesse[1] discusses the **diagnosis of gastric ulcer from disease of the gall-bladder.** He refers largely to the literature, and particularly to Kuttner's discussion of this diagnosis. He then takes up the individual symptoms of ulcer and gall-stone. He finds himself in agreement with many prominent authors in the statement that there are practically no symptoms of either the one condition or the other that can be depended upon as absolutely distinctive. Vomiting is, of course, present in both cases, and while vomiting of blood is usually characteristic of ulcer, it is not by any means usually present, and vomiting of blood may occur in rare instances in cases of gall-stone. Hypersecretion, hyperchlorhydria, and disturbance of the motility of the stomach may all be present in gall-stone. The presence of a tumor is not distinctive, for tumor may be present in ulcer. One point which he considers of considerable importance is the presence of more or less abdominal distention. This is, he believes, an indication of gall-stone which is of some value. Pain is not characteristic always, as gall-stone pain may be in some instances solely on the left side, and the contrary is also true of the pain in ulcer. It is of importance that **gall-stone pain usually appears at night,** a number of hours after the last meal. But this is not always true, and the pain in ulcer may be postponed for a long time after a meal. The condition of the gastric contents is not at all distinctive. As to tumor, he states that a very large proportion of his cases of gall-stone or cholecystitis have no tumor at all that can be palpated during life, and, as previously mentioned, tumors may be present in ulcer. Icterus is usually looked upon as being a very important symptom in the diagnosis of gall-stone. It is, however, a **comparatively rare symptom;** that is, it occurs **in the early stages** of gall-stone in only about 10 % of cases. Finally, Hesse describes 2 cases which showed difficulty in diagnosis. The first was in a woman of 36 who was said to have had jaundice in childhood, and whose symptoms appeared after the birth of her second child. There were indefinite gastric symptoms without pain at first, but with occasional slight chills. Subsequently she began to complain of severe pain in the back, and had several attacks of severe vomiting and diarrhea without pain. She never had jaundice with these attacks. The stomach was slightly enlarged ; there were various tender points, the most painful being below the right border of the ribs in the parasternal line. There was also a tumor in this region. The gastric contents were hyperacid. A diagnosis of relative pyloric stenosis due to ulcer was made, and confirmed by operation. In the second case, a woman of 29, the symptoms began with an attack of vomiting with chills and severe pains in the epigastrium radiating toward the left. Jaundice was said to have been present. Such attacks were repeated several times a year for 4 years. The last attack consisted in vomiting, diarrhea, severe chills, and great pain, the patient being able to lie only on the left side. She had more or less gastric disturbance throughout the attacks. There was a very tender point in the epigastrium slightly to the right of the median line, and another to the right of the twelfth dorsal vertebra. There were, however, no definite physical signs. This patient was believed by Hesse to have cholelithiasis, but others who had examined her had diagnosed gastric ulcer.

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[1] Arch. f. Verdauungs-krankheit., Bd. VII, H. 3.

W. Fleiner[1] discusses the **bismuth treatment for gastric ulcer.** In uncomplicated fresh cases strict dietetic and rest treatment, carried out for several weeks, is sufficient. If, however, on the use of a solid or a mixed diet any notable symptoms occur, bismuth should be used in large doses. The stomach should be cleansed before the bismuth is administered, and the intestine should be emptied by enema. In cleansing the stomach the author advises the use of from 3 to 5 ounces of warm Carlsbad or Vichy water some hours after food has been taken or before breakfast. About an hour later he gives from 75 to 150 grains of bismuth, well stirred in about 3 ounces of water. Half an hour after this a meal may be taken. As a rule, the bismuth should be given once a day. When the symptoms have disappeared for at least a week, the dose should be decreased. With old gastric ulcers which are half scarred over, remnants of food are likely to catch in the irregular ulcer and cause symptoms of irritation. In such cases Fleiner advises washing the stomach and administering from 200 to 300 grains of bismuth through the stomach-tube. The patient should then assume the position which in the individual case seems to be most likely to cause the bismuth to settle upon the ulcer. When pyloric stenosis complicates the condition, the symptoms may be relieved by lavage and the use of bismuth; but gastroenterostomy is usually required in such cases, if there is to be any attempt at an actual cure. Large doses of the drug are **contra-indicated in cases in which there are deep, diverticulum-like projections of the stomach-wall.**

C. Pariser,[2] in some remarks concerning the treatment of gastric ulcer, states that he has used Fleiner's method with much satisfaction; but, because of the expense attached to this method, he has altered it by ordering, instead, **a mixture of chalk and talcum,** in equal parts, giving 60 grams per day, and **adding magnesia usta as an antacid and mild purgative**—the magnesia being given in the quantity found to be necessary. The results, he thinks, are better from the use of this mixture than from that of the bismuth. This treatment is not satisfactory if there is any stenosis. The author has had less satisfactory results from the employment of the silver nitrate treatment. He considers the **dorsal tender point extremely important** in the diagnosis of gastric ulcer and in the determination of the degree of improvement. In diet, he finds that **milk is much better borne if some such sub-**

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[1] Therap. der Gegenwart, No. 11, 1901.
[2] Deut. med. Woch., April 10 and 24, 1902.

stance as finely ground tapioca is mixed with it, as this makes the curd finer. He insists that bouillon must not be given, because it has practically no nutritive value, and the salts and extractives are irritating. Nutrose he considers a very valuable preparation. Rectal alimentation he thinks less of with increasing experience. He believes that the main good that it accomplishes is absorption of water. This is increased by adding some alcohol. He considers, however, that rectal enemas occasionally may be carried, by reversed peristalsis, throughout the whole digestive tract, and mentions the case of a girl who, after each enema containing sherry wine, had marked eructations, with a distinct taste of sherry, and marked gastric pain. These symptoms ceased after stopping the use of the wine.

Rectal Alimentation.—(See also last abstract.) J. Mochizucki[1] has investigated the **possibility of the absorption of albumin through the mucous membrane of the large intestine,** after administering an enema of thymus gland, which was first cooked and then chopped fine. This was moistened, table salt and laudanum added, and administered daily to a person in good health in addition to the food taken by the mouth. It was found that during the period in which this injection was given the uric acid and phosphates of the urine increased to a considerable degree; it was therefore decided that the nucleoproteid was absorbed, and that this was evidence that albumin can be absorbed from the intestines. Further evidence was seen in the fact that the nitrogen of the urine was increased during the same period. [This is not sufficient testimony. The increase in the nitrogen of the urine was comparatively slight, though this does seem to indicate some absorption of albumin, but the fact that the uric acid and phosphates were increased does not necessarily indicate that the albumin was absorbed, as there has been evidence lately from several sources that nucleoproteids are at once broken up into the nucleic acid element and a pure proteid element. Consequently the former might readily be absorbed without any absorption of the latter, and this roundabout method of determining whether there is absorption of albumins is not reliable. Furthermore, the subject, who was supposed to be entirely healthy, was, according to the results given, retaining very large amounts of nitrogen throughout the whole experiment, an observation contrary to normal conditions. It seems questionable whether the technic of the experiment was accurate.]

F. Reach[2] has made a study of the **absorption of carbohydrates through the rectum.** He finds that rectal injections containing 60 grams of cane-sugar or dextrin in 120 to 200 cc. of water, or 100 grams of starch in 300 cc. of water, **do not cause any increase in the respiratory gas-interchange;** while 60 grams of cane-sugar in 120 cc. of water given by the mouth cause distinct increase in the respiratory interchange. The rectal injections do not produce glycosuria or dextrinuria. The method of determining the amount of carbohydrates absorbed by estimating the deficit in the stools is wholly useless, because of the changes that carbohydrates are likely to undergo in the intestines, particularly from bacterial action. A certain amount of sugar is absorbed from sugar injections, but this is **far less than the amount absorbed when given by the mouth.** The absorption is also much slower than by the normal method. In these points Reach directly contradicts a number of earlier writers. His similar results with starch were also contrary to those of other workers. The absorption of starch is slight, and decreases after a short time; so that Leube's statement that the slowness of the absorption of starch from the rectum is compensated for by a long stay in the rectum is not borne out by his experiments. Dextrin was absorbed more slowly but more completely than sugar, and the author believes that this substance may well be tested further in rectal alimentation. It also causes less irritation of the intestine than sugar, and has been used with satisfaction for years in the dextrinized cereal preparations added to the milk-mixtures of infants.

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[1] Arch. f. Verdauungs-krankheit., Bd. VII, H. 2.

[2] Arch. f. exp. Path. u. Pharm., Bd. XLVII Hefte 3 u. 4.

F. Reach[1] **recommends that gelatin be used as a part of nutritive enemas.** He finds that, when injected into the rectum in normal salt solution, this substance is **absorbed as well as albumoses.** His investigations chiefly concern the contention of Grützner that when injected into the rectum foods are absorbed by the higher portions of the intestine. This Reach considers to be incorrect. He carried out laparotomy on dogs, tied the rectum from above, and then administered nutritive enemas into the rectum. The animals were killed from 4½ to 5 hours afterward, and it was found that there had been actual absorption from the rectum.

C. R. Box,[2] in discussing ulceration of the stomach and duodenum, recommends, in cases of doubtful nature, a **strictly conservative treatment.** He insists upon the importance, in doubtful cases, of examining the urine and of determining the pupillary reflexes and the condition of the eyegrounds, with the especial thought of locomotor ataxia, tumor of the brain, and nephritis. If an ulcer is strongly suspected, he insists upon the importance of keeping the patient in bed, and on a strictly limited diet.

Diet in Gastric Disease.—S. Weissbein[3] states that the **staining analysis of nutritive preparations gives very valuable results,** and can be carried out by any practitioner. He used Pappenheim's panopeptic mixture, and proceeded as follows: A small quantity of the substance to be examined was placed in a centrifuge glass about half full of water; 10 drops of the stain were added; the mixture was shaken for 2 minutes and then centrifugated; and was then repeatedly washed by centrifugating until the wash-water was clear. It was then examined microscopically. Albuminates, albumins, and peptones are acidophile; nucleoalbumins and caseins are basophile, as is cellulose. Starches may be stained with iodin, and fats with osmic acid; but neither of them stain with the ordinary anilin dyes.

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[1] Pflüger's Archiv, Bd. LXXXVI, p. 247.

[2] Brit. Med. Jour., Feb. 8, 1902.

[3] Deut. med. Woch., Jan. 9, 1902.

H. Brat[1] has prepared a substance which he calls **gluton,** by allowing acids to act upon gelatin at a high temperature for several hours. The acids are then neutralized; and the preparation is dialyzed or filtered, and dried by evaporation. It furnishes a yellowish-white powder, which does not gelatinize in strong solutions, but which is readily soluble in water. The author states that it **can be used to advantage** as a drink, with some flavoring, or may be added to soft food, etc. He also says that his investigations of metabolism show that this substance is quite as useful as gelatin in **restricting protein disintegration;** and that it is just as valuable as the albumin nutritive preparations, and acts in smaller doses. It is well absorbed and does not seem to cause gastrointestinal disturbances. He believes that it is a useful addition to the dietary. He found no indication in the conjugate sulfates and fatty acids of the urine that it caused any increase in intestinal putrefaction. The excretion of uric acid was decreased, rather than increased, while it was being used.

Frentzel and Toriyama[2] discuss **the value of meat extracts,** and, contrary to the opinions of previous writers on this subject, insist that their metabolism experiments have convinced them that the value of these extracts is great. They state that **63.6 % of the caloric value of these substances is made use of in the organism** for the production of energy. They note that the so-called albumin-free extracts may contain from 10 % to 15 % of albumin.

W. G. Morgan[3] was led by the numerous recent reports concerning the influence of sugar upon metabolism to a determination of the **effect of sugar upon the gastric secretion.** He reaches the conclusion that the use of large amounts of sugar, in normal and abnormal persons, **reduces the secreting power of the stomach,** both acids and enzymes being decreased. This would, therefore, seem to be a good method for treating hyperacidity; in most cases it seemed to be possible to vary the acidity at will, by varying the amount of sugar used.

Massage.—M. I. Knapp,[4] after a study of the **effect of massage upon the motility and chemical functions** of the stomach in several cases, decides that the motility is **decreased rather than increased,** and that the free HCl shows a tendency to diminish rather than to increase; he consequently believes that massage should be used only for its psychic effect, and that only very light strokes should be executed, without any attempt to massage the stomach itself.

Hemorrhage.—W. S. Fenwick[5] notes that he has excellent results in gastrointestinal hemorrhage from the use of **suprarenal extract,** and that there have been no unfavorable effects. The results are most satisfactory in cases of recent gastric hemorrhage; in chronic cases the vessel is often so embedded in fibrous tissue that it is incapable of contraction.

Carcinoma of the Stomach.—W. A. Boekelmann[6] contributes some investigations concerning the **pathologic anatomy of the human stomach** in cases of ulcer and carcinoma **with known chemical and motor function.** The first series of cases were those which had been operated upon and in which portions of the mucosa had been obtained at the time of operation. There were 44 cases, in 38 of which the secretion was well known. There were 17 cases of ulcer; 9 of carcinoma following ulcer, with persisting HCl secretion; and 17 or 18 of carcinoma with very slight HCl secretion or absence of HCl. The **chief characteristics of the cases of ulcer** were that, while the glands were of about normal length, there were no goblet-cells or cells resembling the intestinal epithelium, only slight pouching of the necks of the gland, little or no increase of interstitial tissue; slight small-cell infiltration into the superficial layers; few or no eosinophile polynuclear leukocytes; few or no hyaline bodies; many new blood-vessels and hemorrhages; large numbers of parietal cells, often more than normal, and the principal cells usually seemed to be more seriously diseased than the parietal cells. In the **cases of carcinoma following ulcer** the mucous membrane showed practically the same changes as in ulcer, with the exception of one instance, in which the gland tubules were very unduly prolonged and there was decided increase of the interstitial tissue. The cases of **carcinoma with slight or absent HCl secretion** showed varying conditions. The glands were often unduly long and the tubules abnormally elongated. The number of the gland tubules varied greatly. There was a varying amount of increase of the interstitial tissue, it being sometimes extremely marked and usually decidedly increased. Polynuclear neutrophiles and polynuclear eosinophiles were found, sometimes in very considerable numbers; but the eosinophiles showed no relation to the number of blood-vessels or to the presence of hemorrhage. Hyaline bodies were frequently found in large numbers. There were often many goblet-cells, and frequently numerous cells of the character of those normally found in the intestine. The relative numbers of principal and parietal cells varied. Boekelmann then refers to the literature on the changes in the mucosa, and finally directs attention to the fact that **carcinoma following ulcer seems to be much more frequent than is commonly thought.** At any rate, it is so in the Netherlands, for he reports among the operative cases 9 instances of carcinoma after ulcer, to 17 of apparently primary carcinoma; and in 35 cases not operated upon, there were 26 of primary carcinoma and 9 of carcinoma after ulcer. One of the most striking histologic differences between these two classes of cases is the **relatively good condition of the mucosa in the cases following ulcer,** as compared with those of primary carcinoma. The author then refers to the question as to the reason for the absence of HCl in carcinoma, and inclines to the view that it is largely the result of atrophy, combined with poor appetite and cachexia. [His method of determining that a case was one of carcinoma following peptic ulcer seems to have been a very loose one, and chiefly dependent upon the persistence of free HCl.]

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[1] Deut. med. Woch., Jan. 9, 1902.

[2] Arch. f. Anat. u. Physiol., 1901, Hefte 5 u. 6.

[3] Arch. f. Verdauungs-krankheit., Bd. VIII, Hefte 1 u. 2.

[4] Amer. Med., Nov. 30 and Dec. 7, 1901.

[5] Brit. Med. Jour., Nov. 30, 1901.

[6] Zeitschr. f. klin. Med., Bd. IV, Hefte 1 u. 2.

J. Boas[1] contributes a **study of 141 cases of gastric carcinoma.**

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[1] Arch. f. Verdauungs-krankheit., Bd. VII, Hefte 4 u. 5.

As to the onset of symptoms, he states that while, as a rule, the onset is slow, it is not sufficiently recognized that it **often begins suddenly** when the gastric symptoms have previously been either slight or absent. Such an onset was observed in 39 cases. Another striking form of onset is with decided hematemesis or melena. He has observed 4 such cases in this series. The diagnosis in such cases between ulcerating carcinoma, carcinoma following ulcer, and simple ulcer is extremely difficult. The appetite is usually said to be greatly reduced or absent in carcinoma. This is commonly true, but in about one-third of the cases the author found either normal or increased appetite; and it may simply be said that **the appetite is extremely variable.** He believes that it depends largely upon the care given to the mouth and tongue. The course of the disease largely depends upon the nature and the seat of the tumor. It is well known that different varieties of carcinoma show different courses; it is also readily recognizable that carcinoma of the fundus and of the pylorus have very different courses, the latter usually causing death much more rapidly, and producing severe stenosis and motor insufficiency. **Stenosis, however, is occasionally absent in pyloric carcinoma,** and motor insufficiency is not infrequently present in carcinoma of the fundus. In some cases signs of motor insufficiency appear with astonishing suddenness. The evidences of stagnation also largely depend upon the nature and amount of the food taken; and in determining whether stagnation is present, one should always make a number of tests, as it may be present at one time and absent at another. Boas finds **coffee-ground vomit much more frequent in cases of marked stagnation** with the presence of much lactic acid, while he finds minute hemorrhages and pus much more common in cases without stagnation. He notes one case which is illustrative of the fact that gastric carcinoma may run such a course as to allow the subject to continue at his occupation practically up to the time of his death. Among the complications of carcinoma, the author notes the **frequency of disturbance of the intestines,** which is usually constipation, diarrhea being, in his experience, much less commonly present. Edema and ascites are also occasional complications: edema occurring in the early stages of a suspicious condition being a sign suggestive of carcinoma. He has seen hernia epigastrica altogether in 7 cases; **swelling of the supraclavicular glands he never observed** in the early stages of the disease, and he does not believe that this sign has any value as an early indication of carcinoma. He has in a number of instances observed enlargement of the glands about the umbilicus and actual carcinomatous infiltration of the umbilical region. The glandular enlargement is rare, but, when it occurs, is very suggestive. He found **metastasis to the liver in only 21 cases.** The insignificance of his figures he attributes to the fact that most of his cases were ambulatory. Albuminuria was present only 3 times; glycosuria, only once. He states, however, that he has found **glycosuria or diabetes common in rectal carcinoma.** Tuberculosis of the lungs was present in 5 cases; neurasthenia, in 4 cases. The latter is of importance because it is likely, in the early stages, to lead one to suspect that the condition is purely neurotic. Boas notes a number of cases in which the carcinoma developed on the basis of an ulcer, but he insists that we have no definite means of determining during life that such is the case.

J. Boas and A. Kochmann[1] contribute a further study of **occult gastric hemorrhage,** having investigated, in all, 257 cases of gastric and intestinal disease. The method which they recommend for investigating the gastric contents or feces is to take about 5 cc., add about one-third the volume of glacial acetic acid, shake well, and extract with 5 cc. of ether, avoiding emulsification, or, if an emulsion is produced, adding a few drops of alcohol. A few cubic centimeters of the ether extract are then taken, 30 drops of old turpentine oil and 10 drops of tincture of guaiac are added, and a blue tint is produced. The authors state that the **feces may frequently be examined with profit,** when it is inadvisable or impracticable to examine the gastric contents. They divide the cases into 3 classes. In the first blood is not found; this includes anacid, subacid, and acid gastritis, hyperacidity, and hypersecretion. Of course, if the gastric mucous membrane should be at all damaged by the tube, small amounts of blood would occasionally be found; but such a result is only occasional. In the second class blood is at times found; this includes gastric and intestinal ulcer, pyloric and duodenal stenosis, and benign dilation. In stenosis and benign dilation the authors note that so long as there was no stagnation blood was more or less frequently found; while, with improvement in the stagnation, the blood decreased or disappeared. **In the third class they include gastric cancer only.** In the cases of carcinoma, which were 67 in number, they **found blood constantly,** with the exception of 2 instances; and even though stagnation improved under treatment, blood persisted. They believe that the repeated discovery of blood in the gastric contents or stools is of great importance in the diagnosis of doubtful cases of gastric or duodenal ulcer, the first of which are quite common. A positive result is strongly in favor of ulcer; a constantly negative result is distinctly against ulcer. As to carcinoma, a positive result is not of such great importance; but is, nevertheless, an aid in cases in which there is no stagnation. In those cases in which HCl is absent, but in which there is no stagnation, the **discovery of occult hemorrhage speaks in favor of carcinoma;** while the constant absence of such hemorrhages speaks, with great emphasis, against carcinoma. The authors also state that it is possible, in this way, to discover the occurrence of more or less constant slight hemorrhage, which leads to severe anemia; and to control this hemorrhage.

R. Rencki[2] discusses the importance of **blood-examination in the diagnosis of carcinoma and ulcer of the stomach.** He first refers to the red blood-cells and the hemoglobin, and decides that there is **nothing characteristic in them in either of these diseases,** that both of them may vary within wide limits, and that there may be practically no change in either in the early stages of carcinoma; hence, that they are no help in diagnosis. One interesting observation which he reports was the appearance of **large numbers of megaloblasts in a case of gastric ulcer,** in which hemorrhage had reduced the number of red blood-cells to about 1,300,000, and the hemoglobin to 19 %. The megaloblasts appeared only in the last few days of life. The observation is interesting additional testimony that megaloblasts are not so characteristic of pernicious anemia as many authors still consider them. He states that gastric ulcer, of itself, in his experience, causes no leukocytosis. In 2 cases he found a moderate leukocytosis, but in both of these there was a complicating condition; while in the other instances the count of the leukocytes did not go above 8800. The differential count he also found to be without value in the diagnosis. He then made some investigations concerning **digestive leukocytosis,** first determining that in 12 non-carcinomatous cases digestive leukocytosis was present in 9 and absent in 3, the latter cases being, respectively, one of atony, one of intestinal catarrh, and a normal man. In gastric carcinoma, of which 11 cases were examined, he found 4 instances without and 7 with digestive leukocytosis. He is convinced that the disease of the gastric mucous membrane cannot be considered responsible for the lack of appearance of digestive leukocytosis; in one case, for instance, practically the whole of the stomach was involved by the cancer, and yet digestive leukocytosis was present. He noticed that it was particularly in cases of pyloric stenosis, either with or without carcinoma, that digestive leukocytosis was likely to be absent; hence, he tried giving nutritive enemas in these cases. In one instance he produced a distinct digestive leukocytosis in this way; in two others, a slight decrease in the leukocytes; in one, no change. More distinctive results were obtained, however, by observing cases after operation which had overcome the pyloric stenosis. In 6 cases he regularly found that digestive leukocytosis appeared after the operation. He decides, therefore, that the **absence of digestive leukocytosis is ordinarily due to pyloric stenosis,** although it may, of course, be due to severe intestinal disease or some other similar conditions. He also thinks that the absence of digestive leukocytosis in normal persons is due to pyloric incontinence. The result of this would be that the gastric contents would be passed into the intestine more or less constantly, in small amounts, instead of over a comparatively short period and in large quantities; hence, digestion would be more or less constantly going on in the intestine, and there would be no occasion for any sudden increase in the leukocytes. [The latter explanation is hardly acceptable, as pyloric incontinence would, under normal circumstances, not result in prolongation of digestion, but rather in shortening it.]

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[1] Arch. f. Verdauungs-krankheit., Bd. VIII, Hefte 1 u. 2.
[2] Arch. f. Verdauungs-krankheit., Bd. VII, 3 to 5.

C. Reissner[1] has made some investigations concerning the **chlorids in the stomach-contents in carcinoma and the cause of the deficit in free HCl** in this disease. His method was to determine the chlorids after the manner of Martius and Luttke—also adding another factor by using the following method: A portion of the stomach-contents equal to the other portions taken was exactly neutralized; *i. e.*, the free hydrochloric acid and that combined with albumin was converted into fixed chlorids. The mixture was then evaporated to dryness and burnt to a coal, and the chlorids were estimated. This method was sufficient to eliminate the very volatile chlorin combinations, such as ammonium chlorid, which always makes an error in the results. The chief point in the author's results was to demonstrate that while in carcinoma the HCl is reduced, **the total chlorids of the stomach-contents are not reduced;** they may even be increased. This, to his mind, indicates that the secretion of HCl by the stomach in carcinoma is not reduced, but that the hydrochloric acid is neutralized by some alkaline secretion of the cancer. The difference between carcinoma and achylia was very striking, in the latter disease the total chlorids being found reduced. Applying these results to the explanation of the absence of hydrochloric acid in cancer, he states that he believes there are **several factors in the production of this deficit:** one is reflex paralysis of the secretion of HCl by the continued alkaline secretion of carcinoma; another reduction of chlorids in the tissues, owing to the excessive secretion of chlorids into the stomach; and a third, atrophy of the mucous membrane. The latter factor has, by most recent authors, been made responsible for the entire HCl deficit. It is not, however, a satisfactory explanation, as marked atrophy is often absent. Reissner believes that a carcinoma changes the chemical composition of the gastric contents only when it has ulcerated, and thinks that the reason HCl persists in carcinoma following peptic ulcer is that, under those circumstances, the carcinoma tends to grow into the depths of the gastric wall, instead of ulcerating freely on the surface. He says that his view concerning the alkaline secretion of carcinoma is sufficient to explain the frequently observed early deficiency of HCl, which does not seem to be dependent upon any special seat of the carcinoma, and has even been observed in carcinoma of other organs. It also explains the rapid appearance of atrophy of the mucous membrane, the relatively slight change in the ferments, the frequent long persistence of free hydrochloric acid in carcinoma, the not uncommon sudden disappearance of the free HCl, and the reappearance of free HCl after removal of the tumor.

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[1] Zeitschr. f. klin. Med., Bd. IV, Hefte 1 u. 2.

C. P. Emerson[1] reports some investigations concerning the **extent of gastric digestion in cases of carcinoma of the stomach.** He determined the nitrogen in the substance precipitated by half-saturation with zinc sulfate and by complete saturation, and also the nitrogen in the phosphotungstic precipitate. He finds that the primary and secondary albumoses are present in only about half the amount in cancer that they are in other diseases of the stomach, while the peptones and other substances precipitated by phosphotungstic acid are present in a much larger percentage; that is, **digestion in carcinoma is much more advanced.** This, he thinks, confirms the view of Fr. Müller that carcinoma furnishes a ferment which aids peptic digestion; and he believes that he has demonstrated that carcinoma, when added to artificial gastric juice, causes an increase in the proteolysis.

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[1] Johns Hopkins Hosp. Bull., May, 1902; and Deut. Arch. f. klin. Med.

S. Burt[1] reports a case of **colloid cancer of the stomach and omentum,** in which the **diagnosis was made positive by paracentesis,** small particles of neoplastic tissue being recovered with the fluid. The patient was a man of 43, who was extremely cachectic and who had gastric symptoms, but absolutely no pain. His appetite was good. There was a mass below the ribs, ascites, and edema of the ankles. Postmortem confirmed the diagnosis.

R. Otsuka[2] reports a case of gastric carcinoma which during life **exhibited the picture of anemia and gastric anadenia,** gastric secretion being entirely absent. Postmortem showed a small carcinoma in the pyloric portion of the stomach, but there was also diffuse change in the mucous membrane, consisting in widespread round-cell infiltration and degeneration of the glandular cells, with, in part, entire disappearance of the glands. This case is an exception to the usual rule that in carcinoma the gastric mucous membrane does not show sufficient lesions to explain the absence of secretion.

Myoma of the Stomach.—R. Caminiti[3] reports a case of **pure myoma of the pylorus** originating at the pyloric ring and producing stenosis. It was notable in the case that there was marked dilation with atrophy of the stomach-wall, contrary to the conditions that are usually seen in the dilation of stenosis. This was, perhaps, to some extent explained by the fact that the patient was a sufferer from advanced tuberculosis of the lungs, which had produced severe emaciation and general weakness. The author finds 23 cases of myoma of the stomach in the literature, but only one of these was situated near the pylorus.

Tuberculosis of the Stomach.—Breuer[4] demonstrated the stomach of a patient who had exhibited the **signs of achylia gastrica with curious rises of temperature,** the fever being sometimes continuous and sometimes intermittent, but always being closely related to the presence of stagnating contents in the stomach. The postmortem showed general tuberculosis, with numerous chronic tuberculous ulcers in the stomach. Tuberculosis of the intestine was absent. Breuer believed that the absence of HCl and pepsin was the chief reason for the occurrence of tuberculous infection of the gastric mucosa in this case; and the fever, he thought, was due to the ready absorption of toxic substances through the ulcers.

E. Fischer[5] reports a case that he believes was a tuberculous ulcer of the stomach. It was **treated with tuberculin R,** the patient improving greatly. He states that there were distinct local reactions after the larger injections of tuberculin.

Syphilis of the Stomach.—J. C. Hemmeter and W. R. Stokes[6] report a case of chronic hypertrophic gastritis of syphilitic origin, associated with hyperplastic stenosis of the pylorus. With their case they give a general review of the literature on gastric syphilis. The patient was a man of 24, who had a very definite history of syphilis, with dyspeptic symptoms, vomiting, difficulty in digesting solid food, absence of HCl and ferments, and apparently contraction of the stomach, since it could not be distended to any extent. An operation was undertaken by the Heineke-Mickulicz method. The patient did well for several days, and then developed an abscess of the jaw and died of general infection. The autopsy showed the stomach contracted and much thickened. There was a thick layer of dense, white tissue toward the outer edge of the gastric wall. The pylorus was contracted so that it would not allow the passage of a sound larger than a lead-pencil. In the pyloric region the wall of the stomach was about 1 cm. thick. The inner surface was of a brownish-green color. There were scattered hemorrhages and the surface was very granular. The microscopic examination showed the changes characteristic of syphilis. There was diffuse atrophy of the mucosa and large numbers of embryonal cells were present, these changes involving particularly the pyloric portion of the stomach. There were occasional areas of necrosis. The infiltrating cells had a special tendency to collect about the blood-vessels.

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[1] Deut. Praxis, Nos. 1 and 2, 1902.
[2] Deut. Arch. f. klin. Med., Bd. LX, Hefte 5 u. 6.
[3] Gaz. Hebd. de Méd. et de Chir., No. 89, 1901.
[4] Centralbl. f. innere Med., Mar. 1, 1902, p. 235.
[5] Phila. Med. Jour., Oct. 12, 1901.
[6] Arch. f. Verdauungs-krankheit., Bd. VII, Hefte 4 u. 5.

DISEASES OF THE INTESTINES.

EXAMINATION OF THE FECES.

H. Ury[1] describes some results from the examination of the feces, using a method which was suggested to him by Salkowski, and which had previously been used to some extent by the latter author. It depends chiefly upon the fact that practically all the substances which are secreted into the intestines are in soluble form, while those contained in the food remnants are in insoluble form. Hence, **separating the soluble from the insoluble** would give a rough method of determining **which portions of the feces were derived from the food and which from the intestinal secretions.** One of the main points of the investigation was to determine the amount of nitrogen which was derived from the food, and the amount due to intestinal secretion, a subject which has been much discussed by previous students of this question. Ury **found in the watery extract only from 18 % to 33 % of the total nitrogen in the feces,** a result which corresponds fairly closely with that obtained by Rieder when investigating subjects who were taking practically nitrogen-free food. This result is in direct contrast with the statement of Preusnitz, who considered that under ordinary circumstances the greater part of the nitrogen in the feces is derived from the intestinal secretions, not from food remnants. Of the dry residue Ury found over 14 % in the watery extract, and of the ash he found about 27 % in the watery extract. He also determined how much of the nitrogenous matter of the stools was not soluble in water, but was soluble in alkaline solutions. This nitrogenous matter is derived from the nucleins, and he determined that about 34 % of the total fecal nitrogen is of this character. He also makes the interesting statement that, according to his investigations, the calcium contained in the feces is almost wholly derived from the food; and that consequently the usual teaching, that the greater amount of excretory calcium is excreted into the bowel, is erroneous, and that really the **greater amount of catabolic calcium excreted passes off in the urine,** while that found in the feces is derived from food remnants. The chlorids and sulfates are excreted practically solely through the urine. The feces under normal circumstances contain no ethereal sulfates. One interesting observation was the fact that the total sulfates of the feces do increase decidedly when there is diarrhea, an effect which is in entire accordance with our knowledge that when magnesium sulfate is given by the mouth the excretion of sulfates in the urine is lessened, while that through the intestine increases.

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[1] Deut. med. Woch., Oct. 10, 1901.

P. Cohnheim[1] describes an instrument which he has made **for the purpose of removing directly from the rectum portions of feces,** for the purpose of examining them microscopically for parasites. He states that, in this way, he has often been able to discover parasites when the feces, as passed, appeared to contain none.

GENERAL CONSIDERATIONS CONCERNING INTESTINAL DISEASES.

E. Pflüger[2] contributes a number of articles concerning the **necessity for the saponification of fats before their absorption.** As to Löwi's statement that insoluble calcium soaps are absorbed, and that solution is therefore not necessary, Pflüger replies that the calcium soaps are not absolutely insoluble, that the preparations used by Löwi were probably impure, and that his statement that the soaps are dissolved by the lecithin and cholesterin in the intestinal epithelium is not supported by previous work; for no one has, as yet, found these substances in the intestinal epithelium. In the study of the solvent power of bile for fatty acids, Pflüger states that the presence of sodium bicarbonate in a bile-mixture makes the solvent power for fats much higher than has previously been believed. The action upon various forms of fats, however, is very different in degree. It is probable, also, that the bile acts upon the fat and repeatedly enters into action, because of the so-called circulation of the bile, produced by its resorption and repeated excretion. The explanation of the high absorptive action of bile-mixtures on fatty acids is a difficult one, but it is probable that the intestinal fluids provide unusually satisfactory conditions for this absorption. The **most satisfactory conditions as to reaction** are the presence of neutral soaps and of nearly neutral bile.

O. Simon and T. Zerner[3] report some interesting observations concerning the **digestive properties of the intestinal digestive juices.** The patient on whom their observations were made was a girl of 19 who had had severe hematemesis due to gastric ulcer. She had become unable to retain the nutritive enemas which were first given her, and also vomited everything administered by the mouth. Jejunostomy was therefore made, and the patient recovered good health, being fed through this fistula. The intestinal secretion which emptied itself through the fistula was of a clear golden-yellow color, becoming cloudy after standing. It was alkaline to phenolphthalein. It was found that it digested starches rapidly in its native condition, and when slightly acid, but the action upon starches was entirely interfered with by the presence of as much as 0.13 % of acetic acid. When it was slightly or moderately alkaline, but only when alkaline, it digested albumins completely; when at all notably alkaline, it had no digestive action upon starches. These results are considered in connection with the fact that the contents of the upper part of the small intestine are normally acid, while in the lower part they are alkaline. It seems probable from this fact, and from the results reported by the authors, that the **digestion of starches and albumins takes place in different parts of the intestines.**

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[1] Deut. med. Woch., May 15, 1902.

[2] Pflüger's Archiv, Bd. LXXXVIII, pp. 299 and 431; and Bd. LXXXIX, p. 211.

[3] Arch. f. Verdauungs-krankheit., Bd. VII, H. 3.

J. C. Hemmeter,[1] in a contribution concerning **intestinal antiperistalsis,** states that he has demonstrated that a movement of small particles may occur in man, from the rectum to the stomach; and that this movement is aided by suspension of the particles in physiologic salt solution, and is hindered or entirely interfered with by suspending the particles in weak potassium chlorid or HCl solution. The movement occurs along the walls; and while this upward movement is going on, there is a central downward movement of the fecal masses, which is best determined by observing small colored particles in the intestines of very small animals, such as mice. This antiperistalsis, however, cannot move ingesta in large masses; and is, therefore, **not a sufficient explanation of the digestion of nutritive enemas.**

Kaplan[2] describes a condition which he calls **nervous pseudotympany or accordion abdomen.** This condition is a total or partial swelling of the abdomen that is not due either to distention with gas or to tumor. Narcosis causes the abdomen to collapse without the escape of gas, the swelling again appearing as the narcosis passes off. It may appear very rapidly, and disappear with the same rapidity, spontaneously, or under the influence of suggestion. **Three things are constantly observed:** (1) The increase in the volume of the abdomen to as much as 15 cm., the liver dulness descending. (2) The flattening of the arch of the diaphragm. This is often accompanied with pain in the epigastrium or in the lower abdomen. There is no tenderness. There may or may not be digestive disturbance. The respiration is increased in rapidity. The distention disappears during anesthesia and the conditions approach normal. Circulation is poor. Other hysteric symptoms may be present. (3) The fact that with the disappearance of the distention gas does not escape, either through the mouth or through the anus. This **pseudotympanites may be either general or partial.** In the first case it may resemble the conditions in pregnancy, and feel hard and elastic everywhere. In the partial cases the distention is limited to one or more regions of the abdomen. The conditions may be permanent or intermittent, although in the first case decided variations in degree occur. In the intermittent cases the condition varies at different times of the day, with entire irregularity, although it is usually decreased during sleep. **The onset and the disappearance are usually very sudden.** In one patient the condition had lasted as long as 20 years. In some cases recovery was secured within about 2 months. Of 18 cases, 16 occurred in women. The condition is likely to lead to the difficulties in diagnosis that are met in hysteric distention. Anesthesia is the most important method of diagnosis. The prognosis is good, although the author believes that fatal asphyxia may occur. The best method of treatment is through suggestion.

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[1] Arch. f. Verdauungs-krankheit., Bd. VIII, Hefte 1 u. 2.

[2] Gaz. Hebd. de Méd. et de Chir., 1901, No. 70.

D. Duckworth and A. E. Garrod[1] report a case in which **intestinal sand** was passed, and describe the chemical investigation of this substance. It chiefly consisted of calcium phosphate, although there were slight traces of magnesium and iron. The pigment of the sand was partly due to urobilin. There was also a pigment the nature of which could not be determined. In these cases urobilin seems to be present in considerable amounts; bile-pigments, at most, in traces. The authors decide, therefore, that **the sand is probably formed in a region in which bile-pigment has been transformed** into urobilin; viz., in the upper regions of the colon.

Mosse[2] reports some **experimental investigations concerning lead colic.** He administered lead acetate subcutaneously to animals, and produced symptoms which closely corresponded with those recognized as typical of lead colic. He then examined the **ganglia of the abdominal sympathetic system,** and **found marked changes** in those cells—these changes being more advanced in accordance with the greater severity of the colic. In order to exclude the possibility that the cell changes were the result of the intestinal disturbance, rather than of the lead-poisoning itself, he carried out similar investigations with preparations of senna; and in these cases found normal ganglion cells. But he also administered berberin, which caused severe contractures of the intestine, and found in these instances that cell changes similar to those produced by lead were present.

K. Faber[3] discusses a condition which he terms **intestinal dyspepsia.** He insists upon the fact that symptoms which are apparently due to the stomach are not infrequently due to disturbances of the intestine. In the cases that he reports the main features were prolonged constipation, followed by symptoms that were almost exclusively gastric, and consisted chiefly of cardialgia, eructation, anorexia, nausea, and perhaps vomiting. With these, were more or less striking symptoms of nervous depression, with headache and sleeplessness. Treatment directed to the gastric condition alone was either useless or caused the symptoms to increase; while when food containing large quantities of cellulose was given and mild laxatives were administered, and the treatment was directed solely to the intestinal condition, the patients recovered entirely. The **symptoms present had a tendency to simulate gastric hyperacidity** closely. Faber says that tapeworm is extremely common in Denmark, and is very frequently associated with this same class of gastric symptoms.

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[1] Lancet, Mar. 8, 1902.

[2] Centralbl. f. innere Med., No. 11, 1902, p. 281.

[3] Arch. f. Verdauungs-krankheit., Bd. VIII, Hefte 1 u. 2.

M. Einhorn[1] discusses **sitophobia of intestinal origin.** In this condition the patients have an abnormal fear of taking the proper amount of nourishment, being constantly overanxious concerning the uncomfortable symptoms resulting from the taking of food. There were no actual mental disturbances in the cases described. Two cases are reported in which this occurred after persistent constipation, the patients fearing to take the proper quantity of food, as the intestinal tract seemed to become worse with even small quantities of food. In other cases a simliar condition has arisen as the result of chronic diarrhea, the patients finding that the diarrhea improved somewhat after abstinence, and growing abnormally fearful of taking food. The sitophobia **may be so severe as actually to endanger** the life of the patient, and it is important to treat this symptom, to insist upon proper nourishment, to control nervous symptoms as far as possible by the bromids, etc.

R. Schütz[2] discusses **chronic dyspeptic diarrhea** and its treatment. He describes 10 cases in which, with deficient gastric function, imperfectly digested and partly fermented gastric contents were passed into the intestine. After a considerable period, in such cases, the intestine compensates for the gastric disturbances. Finally, irritation grows great, and catarrh of the intestine appears. The bowel movements then have an acid odor, show gas-formation, and are mixed with mucus. The author advises lavage of the stomach and intestines, restriction of the meat in the diet, and the free use of carbohydrates—chiefly in the form of well-prepared cereals. Eggs, plasmon, beef-tea, etc., may be allowed, but usually only small amounts of milk. The patient should gradually return to the use of meat, first using white meat. Fruits should be used last of all.

W. E. Musgrave[3] gives an **extensive study of sprue in Manila,** from a pathologic and clinical standpoint; also furnishing studies of the blood, of the bacteria present, and of the serum reaction. He decides that sprue symptoms are nearly always found in the presence of other well-known lesions, which may be discovered by proper study. Careful study by modern methods failed to show any additional etiologic factor, and the author believes that sprue **must be considered a symptom-group** occurring in the tropics in chronic diseases, especially those affecting the gastrointestinal canal.

[1] Zeitschr. f. diätet. u. physikal. Therap., Bd. v, H. 3.
[2] Sammlung klin. Vorträge N. F., No. 318, 1901.
[3] Amer. Med., Mar. 8 and 15, 1902.

W. Backman[1] discusses the **effects of various forms of diet upon the putrefactive processes in the intestine,** describing some extended experiments in human subjects when using diets consisting largely of meats, carbohydrates, and fats. He decides that, contrary to the general teaching upon this question, the carbohydrates exert no marked influence upon albuminous decomposition in the intestine, while **fats seem to increase the decomposition.** There is no notable difference between diets containing chiefly animal albumin and those containing vegetable albumin. A milk diet or one composed largely of milk is associated with the least degree of intestinal putrefaction. The degree of putrefaction in the intestine **depends almost directly upon the amount of albumin in the food;** but no direct proportion can be regularly estimated between the amount of albumin and the amount of putrefaction. Granting the correctness of the conclusions, we should be justified in considering that if intestinal putrefaction is to be reduced, we should use a milk diet or a diet consisting chiefly of milk, or that the diet should contain small amounts of albumin, relatively small amounts of fat, and a rather free amount of carbohydrate, in order to provide a sufficient quantity of caloric units. His method of studying intestinal putrefaction was purely the determination of the preformed and conjugate sulfates.

H. Singer[2] has investigated the **effect of aspirin and of ox-gall upon intestinal fermentation** by studying the ethereal sulfates and the indigo of the urine when these substances were administered. He found that ox-gall had no decided action; and that, while aspirin did seem to reduce the fermentation in some instances, in others the sulfates and indigo increased. Hence, no distinct and positive effect could be claimed.

Oppenheim[3] reports some experiments which he carried out on rabbits, in order to determine the **effect of physostigmin upon meteorism.** He found that the drug had a very striking influence, and thinks that under certain circumstances it is wholly capable of reducing meteorism. He insists, however, that it must not be used if meteorism has been present for a long time, as it will be ineffectual. It acts only when the muscle-fibers have not become completely atonic and overstretched. In his experiments it acted most strikingly when an artificial meteorism had been partly relieved by making an artificial anus. The use of physostigmin after this caused a very rapid reduction of the meteorism. The fatal results of the latter he attributes to heart-failure, respiration being uninfluenced until after the heart-action had stopped.

K. v. Noorden[4] recommends the use of physostigmin for intestinal tympany. This substance has been used in veterinary practice for a long time. Von Noorden has found that it yielded good results in many of the cases in human subjects in which he has used it. **He especially recommends it for the tympany of typhoid fever,** when the condition is not very pronounced. He employed physostigmin salicylate in doses of about $\frac{1}{75}$ of a grain, one to four times a day, sometimes giving twice as much as this. If any symptoms of poisoning appear, atropin should be used at once.

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[1] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 5 u. 6.

[2] Zeitschr. f. klin. Med., Bd. IV, Hefte 1 u. 2.

[3] Deut. med. Woch., Mar. 27, 1902.

[4] Berl. klin. Woch., 1901, No. 42.

F. A. Packard[1] suggests that in some cases partial intestinal obstruction and marked meteorism may be due to **a loop of bowel containing fluid and acting like a plumber's trap**—permitting the passage of fluids, but preventing the passage of gas. He reports a case in which such a condition seems to have existed. He also especially directs attention to the use of eserin in acute intestinal distention and in chronic constipation due to weakness of the intestinal wall, describing good results in both conditions. The drug was administered in doses of $\frac{1}{50}$ of a grain or less.

W. Tischer and A. Beddies[2] recommend the use of **honthin as an antidiarrheal drug.** It can be used in large amounts and is not poisonous, and they state that they have had excellent results in diarrheal disturbances of various kinds.

APPENDICITIS.

W. A. Edwards[3] has made a collective investigation as to the probability that rheumatism is active in the production of appendicitis, and decides that our **present knowledge does not warrant the use of the term rheumatic appendicitis.**

A. Huber[4] reports 10 cases which illustrate the **possibilities of error in the diagnosis of appendicitis.** In two of these appendicitis was taken for biliary colic; in a third case the contrary error was made. In a fourth also biliary colic was at first mistaken for appendicitis; but the appearance of icterus soon cleared up the condition. In another case a myoma situated in the right side of the uterus was thought to be an appendiceal mass. The author also reports a case of appendicitis larvata in which a perforative peritonitis occurred, together with bilateral pleurisy, the peritonitis causing very few symptoms, and its relation to the appendix not being clearly recognized. In another case a perforative appendicitis showed an extremely close resemblance to gastric ulcer.

R. Lenzmann[5] reports 2 interesting cases of **latent appendicitis,** both of them occurring in women, and both giving rise to peculiar attacks of colicky pains around the region of the umbilicus and stomach. Physical signs of chronic appendicitis were present, and operation relieved the symptoms entirely. Removal of the appendix also, in both cases, relieved a previously obstinate constipation. The author attributes the symptoms, including the constipation, to reflex irritation.

L. Bourget[6] discusses the **medicinal treatment of perityphlitis;** more particularly, chronic cases. Gastrointestinal disturbances are practically always present. In the diet he recommends a large quantity of vegetables, well-cooked fruit, and small amounts of meat. Hyperacidity of the stomach is often present, and should be carefully treated. The author particularly insists upon the importance of keeping the bowels open. There is too much fear of using purgatives, which do less harm than the persistent constipation of these cases. He also **strongly recommends washing** out the intestinal tract, and, if necessary, the stomach. The fluid may be introduced together with the use of massage, so that it may reach a higher point in the colon. He also advises the application of poultices or leeches over the region of the appendix. Such a treatment he has found to result in a continued improvement.

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[1] Phlia. Med. Jour., May 24, 1902.

[2] Arch. f. Verdauungs-krankheit., Bd. VII, H. 6.

[3] Amer. Med., April 12, 1902.

[4] Correspondenzbl. f. schweizer Aertze, 1901, Nos. 15 and 16.

[7] Deut. med. Woch., April 10, 1902.

[6] Therap. Monatsh., July, 1901.

INTESTINAL OBSTRUCTION.

M. v. Cackovic[1] reports an extremely interesting case of strangulation ileus and peritonitis, with a discussion of the differential diagnosis. A man of 29 was taken suddenly with a sharp attack of diarrhea, followed, a few hours later, by severe cramps in the right lower abdomen, with severe peristalsis. Soon after this there was vomiting, with the signs of complete occlusion of the intestine. At midnight the cramps suddenly ceased; but pain and vomiting persisted, and there was no passage of gas or feces. There was entire anuria. The patient **had previously had an attack resembling appendicitis.** When admitted, the following morning, he was collapsed, with persistent vomiting. The abdomen was hard and scaphoid, and was everywhere tender, particularly in the ileocecal region; and there was a dull percussion-note in the latter region. There was no liver dulness, except a relative dulness at the fifth rib. There was marked dulness in the flanks. There were scarcely any peristaltic sounds to be heard. The patient died soon after. Autopsy showed hemorrhagic fluid in the abdominal cavity, injection of the peritoneum, marked distention of the small intestine, and a **volvulus of the ileum,** 1 meter long, in the lower right abdomen—the affected intestine being dark-blue in color and containing a large quantity of hemorrhagic fluid. The appendix was normal. The case had been considered to be perforation peritonitis, because of the history and of the condition when the patient was seen, and because the sudden cessation of pain indicated perforation. The author notes 2 cases which he had previously seen, in which the **pain ceased suddenly after perforation.** One of them was a case of pyloric carcinoma, and one was an instance of incarceration. This **cessation of pain indicated the overcoming of obstruction** through the occurrence of a large perforation. The cause of the condition in this case was thought to be appendicitis. The rapid collapse and the rapidly fatal issue indicated a perforation peritonitis, as did the small, frequent pulse; the superficial, rapid breathing; the scaphoid, tender abdomen; and the tense muscles; and also the absence of peristalsis and the almost entire absence of liver dulness. The only signs indicating ileus were the large peritoneal effusion, the complete obstruction, and the nature of the vomiting. The occurrence of strangulation was apparently due to the diarrhea, which was accompanied by severe peristalsis, the latter producing the volvulus. The rapid course of the case was considered to be due to an **intense intestinal sepsis,** which occurred the more readily because of the existence of an acute diarrhea that indicated that the intestinal contents were decidedly septic.

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[1] Liecnicki viestnik, 1902, No. 3.

E. Quintard[1] discusses the subject of **spastic ileus,** reviewing the cases reported and describing two of his own, in which there were symptoms of ileus that seemed, from the course of the case, to be purely spastic.

DYSENTERY.

K. Shiga[2] gives a résumé of his studies concerning **dysentery in Japan.** He first describes the morphologic and cultural **characteristics of the dysentery bacillus,** and the effects upon it of various antiseptics, and then describes the bacillus in its relation to the disease. The bacilli are found in the latter half of the first week in almost pure culture. Previous to this time they are found only with difficulty, and not in every case. The higher the situation of the dysentery in the intestine, the more difficult it is to find the bacilli early in the disease. They often disappear as the patient begins to improve, and then reappear with recurrences or relapses. The bacilli are found in the intestine almost solely in fresh catarrhal or diphtheric areas, or in the deeper portions of the ulcers, while on the surface of the ulcers and in advanced lesions colon bacilli and other microorganisms are found almost exclusively. Dysentery bacilli are found in the mesenteric glands often, but Shiga has never discovered them in the liver or spleen. He attempted to find them in 5 cases of parotitis which developed in the course of dysentery, but did not succeed. The urine, milk, and blood are always free from them. As to the **agglutination reaction,** he has found a reaction in dilutions of from 1 to 10, up to 1 to 130 in dysentery. It is, however, not always present even at 1 to 10, and it is found at 1 to 10 in patients with other diseases in quite numerous instances. The dilution in which the reaction will occur is to a very considerable degree **proportionate to the severity of the disease,** except in those cases which are of violent course and go on to a fatal ending, in which it is often weak. The disadvantage about the reaction from the standpoint of diagnosis is that it is usually most marked in the period of convalescence, and, as a rule, does not show itself until the second or third week. When the **agglutination increases in its activity, the prognosis is usually good;** if the contrary is true, it suggests an unfavorable prognosis. The methods of distinguishing the dysentery bacilli are the agglutination test in the hanging-drop with immune serum, cultivating in glucose agar, and cultivating in milk. If agglutination appears at once, if no acid is formed in the glucose agar, and milk is not coagulated, the dysentery bacillus may be considered to be present.

Jäger[3] reports that in an **epidemic of acute dysentery,** which occurred among the soldiers in Königsberg he examined 30 cases, and found in all the instances amebas which corresponded in characteristics with **Amœba dysenteriæ.** Their appearance and disappearance corresponded with similar changes in the symptoms. When injected into the rectum of cats, they produced bloody diarrhea. A patient who had recently come from Russia and who had dysentery showed the same condition. The author believes that this epidemic of dysentery, and some cases, at least, of the disease in Russia and Hungary, are due to amebic infection. He was unable to find the bacillus of Kruse in his cases.

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[1] Phila. Med. Jour., July 20, 1901. [2] Deut. med. Woch., Oct. 24, 1901.
[3] Berl. klin. Woch., No. 36, 1901.

W. Kernig and A. Ucke[1] describe a **series of cases of acute febrile intestinal disturbance** in which amebas were found in the stools. Six cases have been observed altogether in St. Petersburg. The cases ran a course of acute dysentery with tenesmus, mucous, and bloody stools. They believe that amebic dysentery is not rare in St. Petersburg.

L. Ebstein[2] reports a case of acute dysentery in which he found a **protozoon which he considers to have been the specific cause** of the disease in this case. The patient was a girl of 26 who was taken sick in Breslau, and who had apparently not been out of that region. The attack came on suddenly, and was associated with mucous and bloody stools and with tenesmus. There was no evidence of any poisoning, She was given tannin injections (2 %) and calomel, and quite rapidly recovered. The stools contained large numbers of round or oval amebas, usually with eccentric nuclei and one or more vacuoles. They were generally found in the midst of large numbers of bacteria, and they probably had a mucous covering, as the bacteria seemed to be entangled in their covering. Their size varied between 5 and 8 microns. The body of the amebas showed fine granulations. The protozoons grew freely on hay infusion, but no cultures were obtained on alkaline bouillon, glucose bouillon, peptone-glucose bouillon, straw infusion, Heyden bouillon, alkaline gelatin, alkaline glycerin-agar, or acid media. The organisms stained well with the ordinary Ziehl-Nielsen method. They could also be well stained in sections of the hardened stools. Cultures on hay infusion and the stools from the patient were **used in infecting cats.** One animal remained in good health; two others showed pronounced symptoms of acute dysentery, and large numbers of amebas were found in their stools. As the animals recovered, the amebas disappeared. One striking fact was that the **amebas from the cats' stools grew well on Heyden bouillon and glucose agar.** The animals were killed, and were found to have numerous, somewhat undermined ulcerations in the large intestine, but amebas were not found in sections of the intestine.

Treatment.—G. E. Richmond[3] reports a series of cases of dysentery treated by the administration of **sulfur.** This was also used for chronic diarrhea following dysentery, and likewise in probable tuberculous ulceration of the intestine. The results were extremely satisfactory. He gives 5 grains at a dose, combining it with the same amount of Dover's powder if the symptoms are acute.

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[1] Petersb. med. Woch., July 6 and 13, 1901.
[2] Arch. f. exper. Path. u. Pharm., Bd. XLVI, Hefte 5 u. 6.
[3] Lancet, Nov. 23, 1901

Kemp[1] believes that tropical dysentery is **more satisfactorily treated by dieting** than by medication. He does not recommend the free use of either astringents or purgatives. If there is vomiting, he uses ice, and small doses of ipecac combined with nux vomica and chloroform. He first employs albumin water as nourishment; as the patient improves somewhat, he adds meat juice; and then very gradually increases the diet. If tenesmus is not controlled by the application of heat, he applies weak cocain solutions to the rectum, up to the internal sphincter.

A. Nehrkorn[2] describes a case of chronic dysentery in which medicinal treatment had been wholly without effect, and the patient had gradually become seriously ill, the hemoglobin being reduced to 30 %, the red corpuscles to 1,800,000, and the weight to 85 pounds (in a boy of 19 who had previously been well). **Right-sided colostomy was undertaken,** the bowel was washed out through the artificial anus, and the patient began to improve almost at once. Within 3½ months the hemoglobin had increased to 75 %, the weight had reached 125 pounds, the patient had no disturbance of the bowels or of appetite, and the artificial anus was closed. **He remained entirely well.** In order to prevent stenosis after the operation, the round opening in the intestine was enlarged by slitting the intestinal wall vertically and closing the wound horizontally. Nehrkorn believes that colostomy should be looked upon as a means by which such cases can, at times, be greatly improved or, perhaps, cured, when medicinal treatment has proved powerless.

CARCINOMA OF THE INTESTINE.

Zinn[3] presented before the Berlin Medical Society a patient who had for a long time had bloody diarrhea with occasional signs of intestinal obstruction. Examination showed a large **tumor of the sigmoid flexure,** which was readily movable; hence the case seemed to be a very satisfactory one for operation; this was carried out, and showed that the mass was a **cylindric-cell carcinoma.** The diseased portion of the intestine was resected. The case was notable because carcinoma in this situation is usually practically unsuited to radical operation.

SYPHILIS OF THE INTESTINE.

F. Rosenfeld[4] reports a case of **stenosis of the small intestine** of syphilitic origin. Operation was undertaken and confirmed the diagnosis. Examination of the specimen showed that the condition was syphilitic. In another case the same diagnosis was made and the syphilitic character was recognized. Syphilitic treatment produced an entire recovery from the symptoms.

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[1] Brit. Med. Jour., Aug. 24, 1901.

[2] Deut. med. Woch., Jan. 2, 1902.

[3] Centralbl. f. innere Med., No. 11, 1902, p. 280.

[4] Berl. klin. Woch., April 7, 1902.

DISEASES OF THE PERITONEUM, LIVER, AND PANCREAS, ETC.

DISEASES OF THE PERITONEUM.

Micheli and Mattitolo[1] report a series of investigations of **chyliform exudates in which the fat-contents were strikingly low.** For the most part they were exudates which, upon superficial inspection, seemed to be the same as those seen in ordinary chylous ascites. They were opalescent, and looked more or less like thin milk. The opalescence did not disappear through sedimentation, filtration, or centrifugation; they differed from actual chylous ascites only in that they never contained more than 0.15 % of fat—the smallest quantity of fat which, according to Letulle, is necessary to make a fluid appear opalescent. The authors agree with Arcoli in attributing the appearance of such fluids to the **presence of a globulin-like substance,** and state that after precipitating the globulin, the milky appearance of the fluid entirely disappeared. This opalescent appearance due to globulin seems to have no special clinical significance. These exudates are found under the most varied circumstances, and in very different diseases.

DISEASES OF THE LIVER.

H. Strauss[2] reports a method which he believes will be **of value in determining the functional condition of the liver.** He first made some attempts to ascertain the condition of the liver function by determining the amount of volatile fatty acids in the urine; but while he believes that this method may have some value, it was not satisfactory. There have also been some attempts to determine the liver function by comparing the amount of ammonia with amount of total nitrogen. This method was, necessarily, wholly unsatisfactory, as might have been postulated from the beginning. The method which he now reports is the investigation for **alimentary levulosuria.** He gives the patient 100 grams of levulose on the empty stomach in the morning, and investigates the urine in the 4 hours following for the presence of levulose, using Seliwanof's reaction, copper reduction, polarization, and the fermentation test. Of 29 cases of disease of the liver, he found alimentary levulosuria in 26. Of 58 cases in which there was no definite disease of the liver, he found levulosuria in but 6. These latter 6 cases are not sufficient to indicate that the test is a bad one, as all of the patients were suffering from some condition which might readily have produced liver change. A more important observation was the fact that in 10 % of the cases with liver disease the test was negative. Strauss believes, however, that if only positive results are accepted as being of importance, and if it is remembered that no test is pathognomonic of any condition, alimentary levulosuria may be considered a valuable method of investigating the functional condition of the liver. He describes several other

[1] Gaz. degli osped., No. 111, 1901.

[2] Deut. med. Woch., Oct. 31, 1901.

cases which further indicate the value of the test; for instance, in two diabetics that had hypertrophic cirrhosis of the liver there was decided levulosuria with scarcely any dextrose in the urine. The diabetic usually reacts to levulose by showing a dextrosuria. In these cases, therefore, the levulosuria must almost certainly be attributed to the liver disease. In 2 cases of carcinoma of the pancreas with coincident disease of the liver alimentary levulosuria was observed without any dextrosuria. These cases speak in the same way for the disease of the liver as the cause of the levulosuria. Pancreatic diabetes always shows dextrosuria after the use of large amounts of levulose.

J. Bruining[1] has investigated the occurrence of alimentary levulosuria in liver disease. He **confirms the work of Strauss,** and states that in 11 cases of cirrhosis there were 10 that showed alimentary levulosuria. Dextrose was given in 15 cases; the result was negative in 13 and positive in 2, the latter both being cases of cirrhosis. Saccharose was given in 15 cases, 13 of them being cirrhosis; 13 of the 15 excreted dextrose with the urine. The author does not believe that Strauss is correct in thinking that it is the levulose of saccharose that is excreted in the urine, since cases reacted differently to levulose and to saccharose, and the sugar that was excreted was usually glucose. He considers it probable that different cases of liver disease **may show glycosuria after saccharose and no levulosuria after levulose, or vice versa;** and that by this means it may ultimately be possible to determine more accurately the nature of the liver disease.

S. Talma[2] discusses the **bactericidal quality of the bile.** He experimented by operating upon rabbits, doing abdominal section in a normal salt solution bath (which prevents shock), exposing the gall-bladder, and injecting into it various cultures. The chief results of his investigation were to show that the bile contains some substances which interfere with the growth of most of the colon bacilli, typhus bacilli, and diphtheria bacilli injected. The sensitiveness of the different bacilli used varied decidedly. The term virulence cannot be used as synonymous with infectiousness for the bile-passages; the bile had different bactericidal effects at different times and in different animals. One of the most important facts bearing upon occurrence of infection was the number of microorganisms which lodged in the bile-passages, the tendency to infection increasing greatly with the number injected. The epithelium of the bile-passages and the liver-tissues are **decidedly resistant to the invasion of various microorganisms,** particularly the diphtheria bacilli. He noticed in repeated instances that there was a chronic or subacute infectious cholangitis as a result of the infection.

P. Lereboullet[3] states that in the icterus of the newborn the examination of serum always shows the presence of bile-pigment; and that the **jaundice of the newborn is, therefore, hepatogenous.** Bile-pigments are often absent from the urine, but Haycraft's reaction for bile-acids is frequently positive. The absence of bile-pigments from the urine is probably due to their retention, possibly through the action of the kidneys.

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[1] Berl. klin. Woch., June 23, 1902.
[2] Zeit. f. klin. Med., Bd. XLII, Hefte 5 u. 6.
[3] Gaz. Hebd. de Méd et de Chir., No. 93, 1901.

G. Ajello and E. Cacace[1] state that while bile-acids are never found in the urine of man or the lower animals under normal circumstances, they found them **constantly present in 70 cases of hepatogenous icterus.** They likewise found them in the urine of animals after the injection of the salts of bile-acids.

Jaundice.—H. Dufour[2] reports 2 cases of catarrhal icterus, in which, during the course of the icterus, the **cardiac contractions became slow and a pulsus bigeminus appeared.** The author believes that when this appears in icterus, one should suspect that the myocardium is functionally weak. Bile produces this effect by its toxic action upon the myocardium and cardiac nerves; but, since it is a rare symptom, it probably indicates an especial susceptibility of these structures.

H. Benedikt[3] reports 2 extremely interesting cases of **icterus of pregnancy,** which occurred in sisters, one 36 and the other 25 years old. The family history was good. The first patient was married when 25 years old, and in 3 pregnancies had a history of intense itching of the skin, beginning early in the pregnancy, with slight symptoms of disturbed digestion, and soon followed by a marked icterus. In each instance the **pregnancy was spontaneously interrupted;** and the fetus, in one case born in the seventh month, and in another at the end of the seventh month, died soon after birth. The patient's liver was enlarged and somewhat tender; the spleen, decidedly enlarged. The blood-examination showed moderate anemia. The examination of the gastric secretions was negative. The **urine showed no bile-pigments, but a marked urobilin reaction.** There was no increase in the indican, and tests for alimentary glycosuria were negative. After the interruption of the pregnancy the symptoms all disappeared quite rapidly, although her hepatic and splenic enlargement persisted; decreasing, however, considerably. The younger sister had exactly similar symptoms in her first pregnancy; but the latter ended with a normal labor, and the child lived and seemed healthy. In her second pregnancy she had similar intense itching; but she aborted before the development of icterus. **Compression of the bile-duct could be absolutely excluded** in these cases, because of the early occurrence of the jaundice. The other important form of icterus of pregnancy—viz., acute yellow atrophy—was, of course, also readily excluded. There was no evidence of distinct gastrointestinal disturbance in either sister. The development of the itching long before the jaundice spoke rather for a distinct alteration in the liver parenchyma than for a simple stagnation icterus; and this view is supported by the not uncommon occurrence of acute yellow atrophy in pregnancy. The author is inclined to think that in both cases there existed a **slowly progressing cirrhosis,** which was caused to advance rapidly by the occurrence of pregnancy, the cessation of pregnancy causing the disappearance of the increased disturbance. He notes similar instances of acute exacerbations in central nervous disease, particularly multiple sclerosis. It is not improbable that so-called vicarious menstrual icterus is of similar character.

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[1] Giorn. interne delle scienze med., 1901, Fasc. 9.
[2] Gaz. Hebd. de Méd. et de Chir., No. 86, 1901.
[3] Deut. med. Woch., April 17, 1902.

H. Peck[1] reports an interesting outbreak of **epidemic catarrhal jaundice** in Derbyshire, England. The hygienic condition of the region in which the epidemic occurred seemed to be excellent. In all, 69 cases were collected, and probably as many more occurred. The disease appeared gradually, with headache, malaise, drowsiness, abdominal pain, and cramps in the legs; and subsequently usually vomiting, moderate temperature, and chilliness or chills. Jaundice came on several days after the appearance of the first symptoms. Generally by this time the temperature was normal, and, when there were no complications, it did not again rise. The liver was not distinctly enlarged, but was frequently tender. The spleen was often enlarged. Nephritis was present in but one case. Croupous pneumonia complicated the condition twice, and urticaria twice; one patient died with the symptoms of acute yellow atrophy, and proved upon postmortem to have the changes characteristic of this disease. This epidemic differed from those previously seen in having a more gradual onset and comparatively low temperature. The incubation period was about 6 or 7 days. The mode of infection could not be determined. The disease was apparently not due to the ingestion of food or drink, and was probably conveyed from one individual to another. It was apparently carried, in several instances, to a considerable distance; and a note states that Kershaw has found that about **150 similar cases occurred** in the district in which he practised. This district had a population of about 26,000, but most of the cases occurred among the working classes; they, however, were well fed and well housed. Sometimes the disease attacked more than one person in the same house. The temperature in the cases observed by him was commonly about normal. All of them recovered.

E. Woirhaye and P. Caziot[2] report a case of **icterus gravis with acute enlargement of the spleen** in a soldier 22 years old who had the symptoms of acute yellow atrophy and died with eclampsia and profuse hemorrhage from the intestine. The liver, however, during the 12 days' illness enlarged, and at death weighed 2140 grams. Pathologically, the authors found congestion, and inflammatory, degenerative, and necrobiotic changes, with compensatory hyperplasia in some parts. The kidneys showed degenerative changes and congestion. In the beginning of the attack there was complete anuria and marked cardiac disturbance, with icterus, vomiting, and general typhoid state. In the scanty urine of the early part of the attack, the urea, the nitrogen, and the sulfur compounds were greatly diminished. After the seventh day there was a free secretion of urine, and the urea increased; at the same time the heart action became more regular. Both parents of the man were diabetic; and the authors believe that the patient had a congenitally weak liver, that this weakness had been increased by an unhygienic life and diet, and that the attack had been brought on by overstrain and a recent attack of pneumonia. Bacteriologic investigation showed **colon-like bacilli** in the liver, kidneys, and some lymph-glands. The temperature was often subnormal—a condition corresponding with a colon-bacillus infection.

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[1] Brit. Med. Jour., Dec. 7, 1901.

[2] Rev. de méd., p. 484, 1901.

Acute Yellow Atrophy.—A. Thurnwald[1] briefly refers to the occurrence of **icterus in syphilis,** and then reports a case of **acute atrophy of the liver in an early stage of syphilis.** The cases of acute yellow atrophy with florid syphilis were collected recently by Richter, the total amounting to 41; and a number of cases have probably occurred since that time. Thurnwald's patient was an officer of 24, who acquired syphilis in the beginning of September, 1900. He **exhibited secondary eruption at the beginning of the illness** that is detailed. The liver was found somewhat enlarged early in January, 1901; and icterus, which was thought to be of ordinary benign form, appeared. In the latter part of the month, however, the general symptoms began to increase, although the syphilitic eruption had largely disappeared. The patient showed delirium, the liver decreased in size, leucin and tyrosin appeared in the urine, the nervous signs and general prostration increased rapidly, and the patient died on February 4th. The autopsy showed petechias scattered in numerous regions, and the liver weighed only 950 grams; the cut surface showed a dark red, marble-like general surface, with numerous yellow masses scattered about. These small yellow masses proved to be icteric liver-tissue; and the cells, even in these masses, were largely destroyed. Cultures showed the presence of the colon bacterium; but as the autopsy was carried out some time after death, this was probably a postmortem infection. The cause of acute yellow atrophy in syphilitic cases is probably the syphilitic toxin. It occurs much more frequently in women than in men, apparently because the organism of women has less resisting power than that of men.

T. R. Bradshaw[2] reports a case of acute yellow atrophy which had an **interesting clinical history.** The patient was a woman of 24 who had jaundice, impaired vision, and choreiform movements. Her previous history was practically negative, except for a severe fright just before the onset of the attack, which had begun with cold and with gastric disturbance. Two or three days later she became delirious. She afterward exhibited jaundice and marked metrorrhagia. She was admitted to the hospital on May 11th. On this day she had extreme choreiform movements. The next day she suddenly became cyanosed and stopped breathing. Artificial respiration was carried on for 4 hours; but there was no attempt at automatic breathing, and the patient died. At this time liver dulness was not discoverable. The urine did not contain either leucin or tyrosin, and albumin was absent. The case was thought to be acute yellow atrophy of the liver, and upon postmortem proved to be such. Bradshaw also mentions a case which began with acute pneumonia and which was followed by the signs of acute yellow atrophy, but in which a postmortem examination could not be obtained; also another case in which the symptoms were those of acute yellow atrophy and in which on postmortem the liver-cells exhibited the changes found in this disease, but in which during life the liver seemed enlarged, and at postmortem was certainly not reduced in size.

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[1] Wien. med. Woch., July 20, 1901.

[2] Brit. Med. Jour., Nov. 30, 1901.

Von Wieg[1] discusses a case of acute yellow atrophy of the liver **with striking psychic disturbance.** A man of 32 was admitted to the insane asylum with acute suicidal mania. He had severe icterus, pronounced motor excitement, continuous delirium, and occasional twitchings in the extremities; and died in a short time. Postmortem showed acute yellow and red atrophy of the liver. There was chronic gastric catarrh, and it was believed that this had given rise to a bacterial infection of the bile-passages, producing an acute purulent catarrh; and that, from this source, there had been a bacterial invasion of the parenchyma of the liver. Bacteria were found in the spleen, which was acutely swollen. There were skin hemorrhages and fatty degeneration of the internal organs. The marked psychic and nervous disturbances may be **attributed either to the infection or to the interference with the antitoxic function of the liver.** The author believes that both factors were active.

S. R. Dudley[2] reports a case of supposed acute yellow atrophy of the liver in a pregnant woman of 28 years. She had **jaundice, with fever,** and gave birth to a child somewhat prematurely. Two days later she went into coma; afterward she became wildly delirious, and 2 days after this she died, after having had for 24 hours persistent coffee-ground vomiting.

A. H. Lister[3] reports a case of **acute fatty degeneration of the liver in a child of 6 years.** He could find no evidence that the child had taken phosphorus, although the clinical picture was that of phosphorus-poisoning. He considers it very difficult to separate phosphorus-poisoning from acute fatty degeneration, either clinically or pathologically, since acute fatty degeneration is itself almost certainly the result of a poison that acts much like phosphorus. One **must always exclude the possibility of phosphorus-poisoning** before making a diagnosis of acute fatty degeneration.

Cirrhosis of the Liver.—G. Ascoli[4] makes a **distinction between atrophic and hypertrophic cirrhosis** of the liver on the basis of some nitrogen-metabolism experiments. He found, in a case of Laennec's cirrhosis, that with a moderately free use of albuminous food, nitrogenous equilibrium occurred; and that it was even easy to cause nitrogen retention. In Hanot's cirrhosis, however, even with a free use of nitrogenous food, and with a large caloric food-value, there was always a nitrogen loss. He decides, therefore, that in Laennec's cirrhosis the increase in connective tissue is the important matter; while in Hanot's the important point is damage to the liver-cells.

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[1] Wien. klin. Woch., No. 12, 1902.

[2] Brit. Med. Jour., Nov. 23, 1901.

[3] Scottish M. and S. Jour., July, 1901.

[4] Deut. Arch. f. klin. Med., Bd. LXXI, Hefte 4 u. 5.

C. G. Stockton,[1] in a paper on the **circulatory disturbances in hepatic cirrhosis,** insists upon the importance of the **toxemia that results from shutting off the portal blood** from the liver, and states his belief that the toxemia has definite symptoms. He especially refers to the cases of operation for cirrhosis of the liver in which such toxic states have followed. He emphasizes the fact that the blood-pressure in the portal vein is low, and that if it is suddenly raised symptoms of toxemia are likely to occur; and thinks that the symptoms may be promptly relieved by purgation. When the vascular changes and the raised portal pressure that permit of the passage of the portal blood into the systemic veins are brought about gradually, as in cirrhosis, the subject is better able to resist the toxemia and becomes gradually accustomed to it.

H. Curschmann[2] reports an interesting case in a man of 32, who was admitted to the hospital because of **grave anemia following repeated hemorrhage.** The red cells were reduced below 1,000,000; the hemoglobin was 24 %. The diagnosis was difficult. There were no signs and there had been no distinct symptoms of gastric ulcer; and, on the other hand, the patient had no meteorism and no ascites, and consequently, cirrhosis of the liver was questionable. He had, however, a much enlarged spleen, and the liver was somewhat small and hard; and a diagnosis of hemorrhage due to hepatic cirrhosis was made. The hemorrhage was considered to have come from the stomach, in all probability, chiefly because of the character of the hematemesis; the blood was driven out with forcible vomiting, while, in hemorrhage from esophageal varices, the author notes that the blood usually flows from the mouth, rather after the manner of regurgitation than of actual vomiting. Postmortem examination confirmed the diagnosis, the **source of the hemorrhage being found within the stomach,** near the cardia. The absence of distinct signs of portal stagnation in the presence of grave cirrhosis of the liver is not extremely uncommon. Curschmann notes 2 cases that died from hematemesis, in which the physical signs of cirrhosis were absent. Such cases can be explained only on the basis of extensive enlargement of the veins connecting the portal and the general venous system. He notes the presence of varices in the lower part of the esophagus in cirrhosis, and insists that varices situated in this portion of the esophagus are almost exclusively due to chronic disease of the liver and portal system. Enlargement of the veins in the upper part of the esophagus has no such meaning. Of 13 cases of cirrhosis of the liver that had fatal hemorrhage, 12 had varices in the lower part of the esophagus. In only one case were the hemorrhages slight and frequently repeated; in the other instances they were severe. The immediate cause of the hemorrhage was usually not discovered during life; postmortem, ulcers were usually found. **Varicosities of the lower portion of the esophagus** produce, in the author's opinion, absolutely **no distinctive physical**

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[1] Jour. Am. Med. Assoc., Sept. 28, 1901.

[2] Deut. med. Woch., April 17, 1902.

signs. The blood may be purely arterial in appearance, or may be dark; and it may be alkaline, even when it comes from the stomach. The examination of the esophagus with the esophagoscope when this condition is suspected he considers absolutely unjustifiable.

E. Hoke[1] reports a second case of **cirrhosis of the liver with severe anemia,** in a woman of 53, the case closely resembling the previous one, except that urobilin appeared later and the subjective signs were less marked. The case was distinguished from Banti's disease by the absence of ascites and of pronounced primary enlargement of the spleen, and the liver was not atrophied. The author, therefore, calls the case hypertrophic cirrhosis of the liver with severe anemia. In the differential diagnosis from pernicious anemia he notes especially the twice-determined **marked reduction of the nitrogen-content of the red blood-cells** (4.8%, as compared with 5.5% in normal persons). In pernicious anemia von Jaksch found the nitrogen-content increased. The red blood-cells varied between 616,000 and 2,600,000.

T. B. Futcher[2] gives a general discussion of **cirrhosis with pigmentation,** and refers to the case previously reported by Osler as Case I. This patient has been under Futcher's observation repeatedly since the previous report by Osler. He is now 50 years of age, and exhibits the same general symptoms as before; but the bronzing has increased, the liver and spleen are enormously enlarged, he has a purpuric eruption on the legs, and he has grown weaker and thinner, though the urine is free from sugar. The hemorrhagic diathesis in this case is interesting, as it has been observed in 5 of the 24 cases collected by Anschütz.

A. Pfannkuche,[3] in discussing **serous peritonitis and perihepatitis in association with pericarditis and pleurisy,** describes 3 cases, all of which were characterized clinically chiefly by severe ascites without edema of the extremities. They closely resembled so-called pericarditic pseudocirrhosis of the liver. Postmortems were carried out in 2 cases. The liver-capsule was found much thickened; and microscopically, in its neighborhood, there were cirrhotic changes in the interlobular tissue. The author discusses the pathogenesis of the isolated ascites which occurred in these cases. He admits the importance of obliterating pericarditis in its production; but considers that other factors, chiefly pleurisy and peritonitis, and especially beginning cirrhotic changes in the liver, were of marked importance in the production of the ascites.

Rovere[4] gives a discussion of Pick's **"pericarditic pseudocirrhosis of the liver,"** reporting 5 cases, in 2 of which the diagnosis was confirmed by postmortem. He insists that we owe to Pick the awakening of general interest in this condition. As to whether the ascites in these cases is due to cardiac cirrhosis of the liver or to the changes in the peritoneum itself, the author believes that in some cases hepatic cirrhosis causes the actual effusion, but that in the majority of cases it is a combination of cirrhosis and peritoneal disease. The latter fact he believes to be demonstrated by microscopic studies of the liver and peritoneum, and by the specific gravity and albumin-content of the ascitic fluid. In such cases cardiac tonics are usually useless or badly borne. Rovere refers to a case reported by Schiassi, in which, after sewing the omentum fast to the anterior abdominal wall, the cyanosis and dyspnea vanished entirely, and the ascites practically entirely disappeared, the improvement having lasted for as long as 2 years.

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[1] Prag. med. Woch., No. 40, 1901.
[2] Jour. Am. Med. Assoc., Sept. 28, 1901.
[3] Inaug. Diss., Kiel, 1901.
[4] Morgagni, No. 10, 1901.

Bucco and Boeri[1] report a case of **marked enlargement of the spleen with cirrhosis of the liver,** without alcoholic, malarial, or syphilitic history. There was marked ascites, and a caput medusæ was beginning to develop. The urine contained albumin, bilirubin, urobilin, and uroerythrin. Alimentary glycosuria was not present. Talma's operation was carried out, **the omentum being stitched to the parietal peritoneum.** After this the abdomen decreased in size, the ascites grew less, and the caput medusæ was seen to increase decidedly. The amount of urine increased, the uroerythrin, bilirubin, and urobilin decreased, and the amounts of urea and uric acid increased. [This case is additional testimony that the splenic enlargement in such conditions should not be considered to be the essence of the disease. This case was diagnosed "Banti's disease." It seems to have been really cirrhosis of the liver with marked splenic enlargement.]

Treatment.—J. H. Musser,[2] in discussing the treatment of cirrhosis of the liver, states that in the management of the ascites he has found calomel and **Apocynum cannabinum** more useful than other drugs. The latter he gives in doses of from 15 to 30 drops four times daily. In the discussion F. Billings said that he had had excellent results from the use of **ammonia muriate** in hepatic cirrhosis. He believed that it had caused the induration to diminish, and even to disappear. J. H. Herrick had had similar satisfactory results, while W. E. Quine had found it utterly valueless. Herrick referred to a case in which there was a large umbilical hernia which became strangulated. Tapping had repeatedly been carried out in this patient. He was operated on for the hernia. A large amount of ascitic fluid was released at this time, and the liver was found to be typically cirrhotic. After the operation the ascites never recurred. Herrick also referred to a case which he believed to be hypertrophic cirrhosis of the liver, in which **severe chills with fever occurred daily** for many months. Punctures of the liver showed no pus and no microorganisms. There was no reason to think that gall-stones were present, and malaria was absent. Herrick agreed with the French in designating certain cases mixed cirrhosis. R. B. Preble stated that he had often inquired into the presence of **hemorrhoids in cases of cirrhosis,** and that he had reached the conclusion that they are certainly not more common in atrophic cirrhosis of the liver than in patients in general. He mentioned 5 cases in which hemorrhage had occurred from ulcer of the stomach. All were men over 40, all were addicted to the use of alcohol, and all had an entire absence of the ordinary symptoms of gastric ulcer. The difficulty in distinguishing these cases from hemorrhage due to cirrhosis of the liver is evident. G. W. McCaskey noted a case that had been tapped 120 times while under his observation, and probably as many as 150 times before death. G. W. Webster referred to cases in which the fluid accumulated suddenly, and the abdomen rapidly refilled, after the patient was tapped; and the cases rapidly went on to death. He had seen 2 such cases within 2 years. A **thrombosis of the portal vein** was found postmortem in both.

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[1] Rivista clin. e terapeut., July, 1901.

[2] Jour. Am. Med. Assoc., Oct. 5, 1901.

Syphilis of the Liver.—M. Einhorn[1] gives a general discussion of syphilis of the liver with a historical discussion of the literature. He divides syphilis of the liver into: (1) **gummas;** (2) **syphilitic cirrhosis;** (3) syphilis of the liver of either of the above forms, but **accompanied by jaundice,** of either (*a*) acute or (*b*) chronic course. He describes 4 cases of gumma of the liver and 1 case of cirrhosis of the liver, and refers to 2 others. He then describes 5 cases of syphilitic disease associated with jaundice. The symptomatology of the condition consists chiefly in pains, which at times are of a severe, colicky character; general symptoms; digestive disturbance; moderate loss in weight; temporary or permanent jaundice; enlargement of the liver; perhaps gummas; and, later, the general evidences of amyloid disease. Enlargement of the spleen is not constant. The diagnosis can be most readily made in cases of gumma. Syphilitic cirrhosis closely resembles ordinary hypertrophic cirrhosis; but in the latter the history of alcoholism, the later enlargement of the spleen, and the less common occurrence of icterus are factors in differentiation; while the history of syphilis and other signs of its presence, and the early enlargement of the spleen, are important points. It has been stated by some authors that gummas may be distinguished from malignant neoplasms by their less marked resistance. This, Einhorn considers impossible. The **prognosis in a comparatively early stage is very favorable.** Of the 10 cases reported in this paper, only one died; while the others were either much improved or entirely cured.

Abscess of the Liver.—W. Osler[2] reports 5 cases of abscess of the liver and briefly refers to another. He particularly insists upon the latency of the first case, fever, chills, sweats, and leukocytosis being absent until just before the patient was operated upon. He also insists upon the **liability to error in diagnosis.** One case was thought to be malignant disease, and in another the diagnosis was empyema. He especially refers to the condition of the leukocytes, and emphasizes the fact that 3 cases had practically no leukocytosis.

Hydatid of the Liver.—H. Caubet and H. Indet[3] report 2 cases of hydatid cyst of the liver which were interesting because of the occurrence of the **hydatid thrill in one and not in the other,** both cysts having the same situation and much the same peculiarities. In both cases the cyst was intensely distended. In the case in which the thrill was present the cyst contained 1.5 liters of fluid; in the other there was scarcely 800 cc. The authors believe that the occurrence of the thrill is not dependent upon the distention of the sac alone, but largely upon its size. **In order that the thrill shall appear,** the sac must be not only large, but in moderate tension, with only a moderately thick and not tense abdominal wall; consequently, hydatid thrill is rare. The patient in whom the thrill appeared, besides having the larger cyst, had a thin, fairly firm abdominal wall. Thick, muscular abdominal walls are almost certain to interfere with the elicitation of this sign.

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[1] Med. Rec., Aug. 17, 1901.

[2] Med. News, April 12, 1902.

[3] Gaz. des Hôp., No. 105, 1901.

DISEASES OF THE BILIARY PASSAGES.

Italia[1] has made an experimental study concerning the **production of biliary calculi by microorganisms;** infecting fresh, filtered, sterilized bile with various microorganisms. He decides that the **colon bacillus and the typhoid bacillus are specific producers of cholesterin stones,** since they cause precipitation of cholesterin. The streptococcus and the staphylococcus cause precipitation of calcium salts, but not of cholesterin; hence, they are probably but rarely the cause of bile-stones. When colon bacilli are mixed with streptococci and staphylococci, the biochemical effect of the former is much more rapid. Under these circumstances, the calculi are of mixed form; they are then produced from cholesterin, calcium salts, and bile-pigments.

A. Böhm[2] discusses the question of the **occurrence of abnormal intestinal putrefaction with obstruction to the flow of bile.** Some authors think that this does not occur; but Böhm found that the amount of the ethereal sulfates in the urine was increased in 3 cases of catarrhal icterus, the absolute amounts being, in each instance, above the normal. The abnormal values for ethereal sulfates also persisted for some time after the disappearance of the icterus.

Binder[3] discusses the **diagnosis of gall-stones,** particularly the tendency to attribute the symptoms to spasm or catarrh of the stomach, appendicitis, movable kidney, and various gynecologic conditions. He reports 96 cases which he has seen in a practice of 15 years, 52 of the cases having been observed for as long as 10 years; 11 died, 7 were operated upon. In 30 icterus never appeared; in 22 the attacks were usually associated with icterus. Of the 96 cases, 73 were in women, 23 in men. As far as operation is concerned, the author believes that if the mortality of operations can be reduced, it would be wise to recommend early operation, so long as the stones are confined to the gall-bladder and not lodged in the bile-passages.

Karewski[4] reports several cases of **intestinal obstruction due to gall-stones,** with some interesting details, and gives a valuable general discussion of this variety of intestinal obstruction. The diagnosis he considers as always extremely questionable; it is generally made only by operation or by autopsy. The possibility of such a cause of obstruction may be seriously considered, however, when fecal vomiting comes on very early in the case and is associated with the passage of feces and flatus for some time afterward, this **peculiar combination of symptoms being due to the very high seat of the obstruction.** It is further of importance that the general prostration, etc., commonly seen in other forms of intestinal obstruction, are absent in the early stage of this variety; the meteorism, also, is less marked—indeed, at times, absent, and the abdomen may even be concave. Later in the attack the condition, of course, exhibits the characteristics always seen when there is peritonitis from any cause; and the diagnosis then can scarcely be even suspected. One interesting characteristic of gall-stone obstruction is that not infrequently the **signs of obstruction appear, disappear, and reappear,** and perhaps go through this course repeatedly, the stone lodging in various parts of the gut without causing absolute and permanent obstruction. This is chiefly due to the fact that the obstruction is often not a purely mechanical one, due to the stone itself, but is the result of the irritation caused by the stone and of the reflex spasmodic contraction of the gut. Operation is always indicated in these cases, unless mechanical and physical methods cause rapid improvement.

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[1] Riforma med., No. 145, 1901.
[2] Deut. Arch. f. klin. Med., Bd. LXXI, Heft 1.
[3] Berl. klin. Woch., No. 7, 1902.
[4] Deut. med. Woch., Mar. 6, 13, and 20, 1902.

F. König[1] reports a very interesting case of **spontaneous rupture of the calculus-containing gall-bladder into the free peritoneal cavity,** in a woman of 70. The absence of previous history of gall-stones and the conditions present made the diagnosis of gall-stones impossible. The patient was *in extremis*, and operation was undertaken. Bile and biliary calculi were found in the peritoneal cavity. Perforation of the gall-bladder was discovered; and, since this organ was very necrotic, it was completely extirpated. The abdominal cavity was cleansed and the wound was entirely closed. The patient recovered without complication. The literature concerning this condition is discussed, and König expresses his belief that changes in the wall of the gall-bladder may be considered to be always present in such cases. In his belief, the result of the perforation is often only a mild inflammation of the peritoneum, unless a virulent infection is carried into the cavity. Even in the former case, however, unless operative interference is carried out, the stagnation of the peritoneal contents and the interference with absorption due to the inflammation cause the bacteria to increase in virulence; and **practically all cases end in death unless operated upon.** He recommends total extirpation of the gall-bladder in most cases, as he thinks that this is safer than to attempt to close a diseased gall-bladder. In the latter case the immediate results are likely to be unfortunate, and a subsequent perforation is not improbable.

DISEASES OF THE PANCREAS.

P. Lazarus[2] contributes an experimental and anatomic study concerning the **pathogenesis of pancreatic cysts.** He made various attempts to produce these cysts in dogs, and has studied 10 cases of cysts in the Vienna Pathological Institute. He found that tying the main duct of the pancreas or its larger ramifications does not cause any notable cyst formation. Lobular stagnation of the secretion produces a localized proliferation of the connective tissue, particularly about the walls of the ducts. Tying off a portion of the gland produced a cirrhosis about the ducts, with atrophy of the parenchyma and slight dilation and proliferation of the ducts. A complete obstruction of the outflow of secretion caused hemorrhagic pancreatitis with necrosis. Injection of pancreatic secretion or of irritating substances, also wounds of the gland, cause fat necrosis. Trauma readily produces hemorrhages, which are usually rapidly absorbed; although there may be a local proliferation of connective tissue, which may produce a local stenosis of ducts, and this may produce stagnation of secretion, with dilation and cyst formation; or, after stagnation of the secretion of the gland, a local necrosis may develop, and softening in the center of such areas may lead to cyst formation. Also trauma of the pancreas may lead to cyst formation if the collecting secretion digests the hematoma, the damaged tissue-remnants, and the parenchyma; and if, at the same time, in the periphery, the reactive inflammation is sufficient to produce a cyst capsule.

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[1] Deut. med. Woch., Feb. 13, 1902.

[2] Zeitschr. f. Heilk., Hefte 6 u. 10, 1901.

Rozanek[1] discusses **acute hemorrhagic necrosis of the pancreas and its relation to the climacteric.** He describes the case of a woman of 53, who, during the climacteric, was taken with severe metrorrhagia, and had attacks of extreme pain in the epigastrium, associated with vomiting. One of these attacks, which was particularly grave and had persisted for 5 days, led to collapse and death. There was marked tenderness in the epigastrium, and sugar appeared in the urine. The autopsy showed hemorrhagic pancreatitis. The author refers to the experimental work showing the influence of the injection of acids into the duct of Wirsung, and states that he believes that the **climacteric leads to hyperacidity of the stomach and excessive contractions of that organ,** and that, as a result, there is likely to be entrance of strongly acid gastric juice into the pancreatic duct.

H. W. Allingham[2] observed a case of **acute hemorrhagic pancreatitis** in a man of 26, who had had a similar but milder attack the year before. He had the usual symptoms of acute pancreatitis, but the cyanosis of the face and abdominal walls described by Halsted was absent. There was no icterus. Laparotomy was undertaken because of a suspicion of intestinal obstruction. Death occurred after 48 hours. Autopsy showed fat necrosis. Cultures from the pancreas showed colon bacilli.

G. B. Hunt[3] describes a case of acute hemorrhagic pancreatitis in a man of 56, death occurring within 20 hours. The condition was suspected, but laparotomy was carried out because it was believed that a duodenal ulcer was present and had perforated. There were no signs of peritonitis, except the severe collapse, the **only noteworthy physical**

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[1] Prag. med. Woch., Nos. 28, 39, 41, and 45, 1901.

[2] Lancet, Oct. 19, 1901.

[3] Lancet, Oct. 19, 1901.

sign being the increased dulness on both sides of the abdomen, which was movable. Autopsy showed acute pancreatitis, with widespread fat necrosis.

M. Leonhardt[1] reports a case in which digestive disturbance, colicky pains, and icterus caused a suspicion of cholelithiasis. A tumor which did not move with respiration then appeared. It lay to the right of and above the umbilicus, but was too far to the left to correspond with the position of the gall-bladder. It was, therefore, **thought to be a tumor of the duodenum or of the pancreas.** The suspicion of pancreatic disease was increased by the presence of fatty stools and of sugar in the urine. The further course of the case suggested infection, because of the fever, the rapid loss of strength, the increasing stupidity, the tarry vomit, and the bloody diarrheal stools. The man died about 2 weeks after the onset of his illness. The postmortem examination showed **a duodenal ulcer,** which had become adherent to and caused **infection of the head of the pancreas.** There were widespread hemorrhages in that organ and in the subperitoneal fatty tissue. The pancreas was almost entirely transformed into fatty tissue, and there was necrosis of the slight remnants of the parenchyma. In the neighborhood there were inflammation and numerous fat necroses. Cultures showed staphylococci, streptococci, and two forms of bacilli. The staphylococci alone were pathogenic. The author considers that the changes in the subperitoneal fat-tissue and the atrophy of the pancreas were old, and that then the infection of the pancreas through the duodenal ulcer had produced the symptoms through the consequent necrosis of the slight remainder of the pancreatic tissue. The **swelling of the head of the pancreas constituted the tumor** and produced the obstruction to the outflow of bile. Death was evidently due to septic infection, and, the author believes, not to the action of the pancreatic secretion. He thinks that it is highly probable that in all cases the main cause of fat necrosis is microorganismal infection.

GASTROINTESTINAL AUTOINTOXICATION.

D. L. Edsall[2] reports a remarkable case of **coma, apparently due to acid intoxication sui generis.** A man in fairly good health was suddenly overtaken by coma which lasted 10 hours, during which time he presented no signs of paralysis, but marked cyanosis, weak circulation, and a strong odor of acetone, together with acetone and diacetic acid in the urine. He recovered completely and very rapidly. He was treated with stimulation and intravenous injections of sodium bicarbonate. He never had any signs of diabetes. The condition of nondiabetic acid intoxication is discussed, and the attack in this patient is attributed to acid intoxication.

A. B. Duprey[3] reports 2 cases of sudden **coma, apparently of intestinal origin,** since recovery was rapid and complete after free purgation.

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[1] Virchow's Archiv, Bd. CLXII, Heft 2.
[2] Phila. Med. Jour., June 28, 1902.
[3] Lancet, June 28, 1902.

He also mentions the case of a child of 6 who was subject to constipation, and who dropped dead while playing. No cause for death could be found, except that the descending colon was jammed full of feces.

DISORDERS OF THE URINE AND DISEASES OF THE KIDNEYS.

GENERAL CONSIDERATIONS CONCERNING RENAL DISEASE.

H. Senator[1] discusses at some length the recent teaching of Israel and his followers concerning **renal colic, renal hemorrhage, and nephritis.** He leaves out of the discussion purely surgical questions. As to the statements that the urine may be free from albumin and casts in severe nephritis, that the urine may contain casts when albumin is absent, and that severe hemorrhage may occur in paroxysms in nephritis, he admits their truth, but considers that these statements are not new, and have previously been generally known. He **does not admit the correctness of the statement that there may be unilateral nephritis,** if this means that there are unilateral cases of Bright's disease. He then discusses the following statements: "Renal colic may be produced by nephritis, and this may closely simulate the colic of renal calculus"; "there are cases of bilateral nephritis in which unilateral colic occurs"; "nephritic hemorrhages may occur with or without attacks of colic"; "the hemorrhage is not the cause of the colic, both phenomena being the result of congestion of the kidney"; "a large number of the cases which have previously been known as nephralgia, hematuric nephralgia, or angioneurotic renal hemorrhage, are due to nephritic processes"; "incision of the kidney has, in many cases, a favorable effect upon the nephritic process and its symptoms." Senator goes into a careful discussion of the cases reported by Israel, and decides that in 11 of the 14 there was no evidence of tension and congestive swelling, and that the result, consequently, **could not have been due to a relief of tension.** As to the presence of nephritis, he believes that in 7 of the 14 cases it was not shown to be present; that in 2 it was present, there being a rapidly fatal issue; and that in 5 it was apparently present, and recovery from operation occurred. He insists, however, that even if a small portion of the kidney does show the presence of nephritis, or if nephritis is actually accepted to be present, colic and hemorrhage cannot properly be attributed to the nephritis. The results of the operation, when favorable, have, he believes, usually been **due to a breaking-up of adhesions,** which have caused traction and torsion of the kidney. Such adhesions were present in 9 of Israel's 14 cases. Incision of the kidney, Senator believes, is useful in cases of anuria in which there is very marked increase of tension in the capsule. It **does not act by relieving the congestion,** however, **but by furthering the flow of urine.** He thinks that one **should not attribute hemor-**

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[1] Deut. med. Woch., Feb. 20, 1902.

rhage to inflammation, but should rather consider that in such cases undiscovered small calculi or gravel, tubercle or other growths, aneurysmal or varicose changes in the vessels, hemorrhagic diathesis, etc., have been the cause; or, if these are absent, that the hemorrhage is neuropathic and due to vasomotor disturbance. Senator does not believe that incision of the kidney is a proper treatment for hematuric nephralgia. He admits, however, the diagnostic importance of the procedure.

J. Israel[1] replies to Senator's criticism of his teaching concerning renal colic and renal hemorrhage, and their relation to nephritis. He goes into extensive details in proof of his previous statements, and **insists that renal colic and renal hemorrhage may be due to nephritis;** that there may be a unilateral nephritis; that incision of the kidney does good; and that it apparently does this by relieving congestion.

Fiori[2] has made a study of **functional hypertrophy of the kidneys,** from an experimental, a clinical, and a histologic standpoint. Removal of one kidney causes interstitial and, subsequently, parenchymatous changes in the other kidney; and these may sometimes ultimately produce a fatal issue. The renal changes are due to mechanical disturbance of the circulation and increase in the toxic substances which the organ must excrete. The increase in the size of the kidney may be as much as 60 %, and it usually appears within from 3 to 3½ weeks after the removal of the other kidney. The increase in size is indirectly proportional to the amount of histologic change. Functional hypertrophy may be due to increase in the size of the epithelial elements or to their proliferation. The proliferation causes the formation of strands of epithelial elements in the peritubular connective tissue, which causes the condition to resemble the embryonal state of the organ. The author thinks **that actual new canals are never produced.** One can not only remove one kidney from dogs without killing them, but can likewise extirpate a half to two-thirds of the remaining kidney, only 1.12 to 1.20 grams of kidney substance per kilo of animal being necessary to preserve life.

Dell' Icola[3] has made a study of **digestive ferments in the urine in normal and pathologic conditions,** chiefly for the purpose of attempting to determine whether these ferments are purely derived from the digestive organs or whether they reach the blood from other sources than the glands of the alimentary tract. In febrile diseases and other exhausting conditions he found that the amount of the ferments in the urine always **corresponded inversely with the general condition and with the power of the organism to react.** He emphasizes the fact, however, that in severe disturbances of the nervous system, of various kinds,—particularly after convulsions, with loss of consciousness,—the ferments decrease markedly in quantity, and sometimes disappear entirely. He believes that this indicates that they are **not derived solely from the organs of the alimentary tract.**

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[1] Deut. med. Woch., Feb. 27, 1902.

[2] Il Policlinico, July and Aug., 1901.

[3] Morgagni, Dec., 1901.

METHODS OF EXAMINATION.

K. Assfalg[1] reports his results from the examination of a large series of cases, for the purpose of testing **the value of methylene-blue as an indicator of the functional condition** of the kidneys. He gives an extended description of the excretion of the dye itself, its chromogens, etc., and reports his results from the investigation of 40 cases. He also includes in his conclusions 6 cases previously reported by Strauss. The nature of the cases was chiefly as follows: **chronic interstitial nephritis;** others in which there was suspicion of beginning disease of the kidney, because of lead intoxication, although albuminuria was absent; instances of alcoholism, cardiac disease, etc.; and 5 instances of **traumatic neurosis.** The latter cases were especially interesting because it had before been observed that these patients did not show normal excretion of methylene-blue. Assfalg confirms these observations, and finds that the appearance of the dye is likely to be delayed and its excretion prolonged beyond the normal period. As to the general results of his work, he decides that the hope that this method would be generally useful for testing the function of the kidney has not been fulfilled by investigation; but that **the test has some value, if properly inspected.** It is necessary, however, to study it further before we can definitely say what its value is. We can say with certainty that **an anatomic diagnosis cannot be made from the use of this test.** It may be compared to the determination of the HCl in the stomach-contents in this way: it shows a functional disturbance; not an anatomic change. **It is possible, however, that it may be a fair indication of the existence of interstitial change** in the kidneys.

G. Rosenfeld[2] refers to his earlier papers concerning the diagnosis of cystitis and pyelitis, in which he stated that an **acid reaction of the urine usually indicates pyelitis;** that the **presence of white or red blood-corpuscles which are of irregular contour** also indicates pyelitis, while well-formed blood-corpuscles, either white or red, indicate cystitis; that a **high percentage of albumin** as compared with the amount of pus indicates pyelitis, while the contrary is found in cystitis. If the urine, after sedimentation, contains more than 0.15 % of albumin, it speaks for pyelitis, more particularly if but small amounts of pus are found; while **if 0.1 % or more of albumin is present, pyelitis must be considered probable.** The author then refers to a case of hydronephrosis in a man of 37, in which operation was performed and clear urine was removed from the sac. For a few days after the operation, however, a gauze drain was left in the wound. The urine during this time was acid, contained leukocytes of ameboid and irregular shape, and also some pale and irregular corpuscles. There was 0.175 % of albumin, while the sediment was but small in amount. These are exactly the conditions which had previously been described by Rosenfeld as indicating the existence of pyelitis; hence, he considers this case to be **experimental evidence of the truth of his statements.**

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[1] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 3 u. 4

[2] Centralbl. f. innere Med., Feb. 22, 1902.

R. Knapp[1] has made a study of the **method devised by Grosz for staining urinary sediments.** That author stated that a 1 % solution of sodium alizarinsulfonate produces characteristic differential stains of the morphologic elements of the urine and of the mucus derived from different portions of the urinary tract. After studying 62 cases, Knapp states that the conditions are complicated, and that it is **not possible by such a study to determine the seat of the disease** with any exactness. He finds, however, that mucus from the bladder takes a red color, and is found in plate-like, homogeneous form, or in finely granulated masses; while that found in cases of disease of the kidney or of its pelvis shows no color, or only a faint yellow stain, and is moss-like or finely striated. In cases of dilation of the ureter or of hydronephrosis the mucus may take a red stain. Casts usually stain yellow; sometimes faint violet. As to the leukocytes, the author finds that those from the pelvis of the kidney are stained yellow, as a rule, and are found in a mass of unstained or faintly yellow mucus. Some leukocytes do not stain, particularly at the height of the disease, and subsequently these leukocytes are not found. It is probable that those which refuse to stain have a more marked vitality; and perhaps **their disappearance may mean that the disease is decreasing in severity.**

Gumprecht,[2] in discussing the preservation of sediments, states that gastric contents may be readily preserved by merely pouring formol over them, and that stools are well kept by preservation in alcohol. He has attempted to preserve leukemic blood, but without success. Bone-marrow provides a brilliant method of demonstrating Charcot's crystals, if first centrifugated in water or salt solution to free it from fat; it may then be preserved in alcohol. As to urine, if only crystalline sediments are desired, these are **centrifugated off and preserved in chloroform.** The **rarer forms of triple phosphates may be artificially produced by adding ammonia to urine.** Calcium oxalate is very difficult to preserve; the same is true of uric acid. **Casts and epithelium may be preserved by centrifugating, decanting the overlying urine, and adding formol.** If often shaken, the whole sediment is well preserved for a long time; if not disturbed, however, the sediment forms an unrecognizable mass, in which one finds some well-preserved casts and cells. The author commonly uses 1 % to 2 % formol. The sediment should not be centrifugated at a high speed.

Fittipaldi[3] recommends the investigation of urinary sediments by centrifugating, after adding 0.75 % sodium chlorid solution, and the same strength of alcohol. **He uses fractional centrifugation,** and states that mucus and pus are precipitated first, then tube-casts, and finally the other formed elements. He says that this method makes it much **more readily possible to diagnose the existence of a nephritis when purulent pyelitis is present.**

Flora C. Fuhs[4] describes the following **test for albuminuria:** 2 cc.

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[1] Centralbl. f. innere Med., Jan. 4, 1902.

[2] Centralbl. f. innere Med., April 19, 1902.

[3] Gaz. degli osped., No. 108, 1901.

[4] Med. News, Mar. 8, 1902.

of equal parts of carbolic acid and glycerin are placed in a test-tube, and 2 cc. of filtered urine added. These are mixed. If turbidity occurs, albumin is present.

DISORDERS OF THE URINE.

Albuminuria.—G. Rem-Picci[1] investigated the condition of 35 young men **after the use of cold baths.** The baths were taken in the open air and were of about 3 minutes' duration, and the temperature of the water was in the neighborhood of 65° F. **Albuminuria was practically a constant phenomenon after such baths,** but was always slight. The less robust and more tender subjects showed the most marked albuminuria. When the temperature of the water was above 70° F., no albuminuria appeared, even after prolonged bathing. It appeared soon after the bath, rapidly disappeared, and was not present the next day. It **occurred even when the subjects were constantly in the habit of using cold baths.** The substance found was serum-albumin. Hyaline casts were also occasionally discovered. Diuresis was often observed, and the total solids were increased. Urobilin was never seen. The author attributes the albuminuria to reflex action due to the shock of the cold water, the action being both upon the vaso-motors and upon the nerve-supply of the kidney. **If such albuminuria persisted, it would contraindicate cold baths.**

L. B. Mendel and D. R. Hooker[2] report a case of **cyclic albuminuria** in a student, who accidentally discovered the albumin in his own urine. Casts and other formed elements were absent. The morning urine was free from albumin. There were two maximal periods of excretion: one at about mid-day, and the other toward evening. **Diet had no influence upon the excretion, nor did muscular exercise.** A horizontal position always caused the albuminuria to decrease.

Graziani[3] discusses psychic and physiologic albuminuria, and decides that there is **no such thing as actual physiologic albuminuria.** He considers that the appearance of albumin in the urine is always an indication that the function of the kidney epithelium has temporarily or permanently suffered; and that if this disturbance of function continues, it will become an actual organic disease.

W. E. Huger[4] describes 2 cases of cyclic albuminuria, with careful studies of the conditions of the urine. He decides that **cyclic albuminuria is due to inflammatory and degenerative changes in the renal structure, to alterations in the quality of the blood, or to alterations in the blood-pressure.** He agrees that a certain number of the cases develop continuous albuminuria and the evidence of Bright's disease, while the vast majority get well. He thinks that continuous rest in bed should always be urged. He believes that **casts will be found in a large percentage of the cases** by very careful examinations, and that, consequently, these are not of such serious prognostic importance in these cases as is often believed.

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[1] Il Policlinico, Aug., 1901.

[2] Jour. Exper. Med., vol. v, p. 647.

[3] Gaz. degli osped., No. 105, 1901.

[4] Johns Hopkins Hosp. Bull., April, 1902.

Albumosuria.—T. R. Bradshaw[1] states that he observed a clear case of Bence-Jones albumosuria in a physician, and was **able to determine the conditions present from the beginning of the patient's ill health.** He found that **albumosuria was the first symptom of the disease,** and was observed for many months before there were any other indications of skeletal changes or of failing health. At that time the quantity was minute, but it increased to 10 parts in 1000. Cases are not likely to be examined at such an early stage, however; and usually, by the time they come under observation, the amount of albumose will be large, and there will be other symptoms.

Donetti[2] reports, under the heading of Kahler's disease, a case in which Bence-Jones bodies appeared in the urine in association with severe pains in the bones, softening of the ribs, spontaneous fractures, distortion of the spinal column, increasing muscular weakness, and emaciation. The patient also had marked thirst and increased secretion of urine. Of interest is the observation that in the early stages of the disease there were **distinct evidences of parenchymatous nephritis, which afterward disappeared.** There were also slight febrile attacks, and the spleen was enlarged; and the author is **inclined to believe that the condition is an infection.**

J. A. Blair[3] reports the case of a woman of 64 who was seen **in an attack of diphtheria.** A routine examination of the urine showed the presence of albumose. The attack of diphtheria was followed by repeated attacks of vomiting, and there was probably marked enlargement of the liver. The patient disappeared from observation after 10 weeks. Albumose had been found each time the urine was examined; it was apparently present in large amounts, the test used being precipitation in the cold with nitric acid andere-solution on heating, with reprecipitation on cooling. [Albumoses are frequently found in small amounts in diphtheria.]

H. O. Mosenthal and W. J. Gies[4] have studied the **usefulness of Freund's method** for demonstrating so-called peptones in the urine, and decide that it is not a differential process and cannot be safely applied to the urine or feces. It reacts to peptones, proteoses, and gelatins, and probably also to seromucoid.

Peptonuria.—M. Ito[5] states that he determined the presence of **true Kuhne's peptone in several cases of pneumonia,** before and after the onset of resolution; also in a case of phthisis, and in the urine of a puerperal woman. His method was precipitation with ammonium sulfate in an acid, next in an alkaline then in a neutral solution, precipitating the filtrate with tannin, with the addition of baryta-water; then obtaining the biuret reaction.

Alkaptonuria.—A. E. Garrod[6] contributes some interesting observations concerning **congenital alkaptonuria.** He states that he has been able to determine that **11 cases of this occurred in 4 families,**

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[1] Brit. Med. Jour., July 13, 1901.

[2] Rivista crit. di clin. med., No. 46, 1901.

[3] Brit. Med. Jour., Sept. 14, 1901.

[4] Amer. Med., Mar. 8, 1902.

[5] Deut. Arch. f. klin. Med., Bd. LXXI, Heft 1.

[6] Centralbl. f. innere Med., Jan. 11, 1902.

and that these cases constitute almost all those that have been observed in Great Britain. He notes, in detail, the following facts: 2 children under his own observation were alkaptonuric, and were born of parents that were first cousins. Pavy likewise stated to him that in a family of 14 children, in which there were 4 alkaptonurics, the parents were also own cousins. He was also told by Kirk of a family containing 3 alkaptonuric children, the parents in this instance, too, being first cousins. In the latter family the mother died, the father remarried, and the one **child of this later marriage was free from alkaptonuria.** Two other instances of this anomaly have come under Garrod's observation, in which the parents were in no way related. In consideration of the fact that alkaptonuria is extremely rare, and that children of own cousins form but a small part of the total population, the **relation between family intermarriage and alkaptonuria cannot be purely accidental. It speaks strongly against an infectious origin** of alkaptonuria, and indicates rather that it is a **chemical anomaly analogous to the common anatomic anomalies.** It is wholly improbable that its occurrence in families is due to the increase of a family tendency through intermarriage, for in none of these instances described was there any history of alkaptonuria in the parents or in other generations. The author also describes a case in which a child in an alkaptonuric family was carefully watched from birth on, to see when the alkaptonuria appeared. The first pigment was observed 53 hours after birth; and from that time on, constantly. **Its first appearance was shortly after the child obtained the first milk from its mother's breasts.** This relation between the beginning of the alkaptonuria and taking nourishment into the digestive tract was to be expected, since he considers tyrosin to be undoubtedly the mother-substance of the homogentisic acid.

F. Mittelbach[1] contributes an observation concerning alkaptonuria. The patient, a man of 44, excreted about 4½ grams of homogentisic acid daily. The method of estimation used was titration with silver. The amount contained in the night urine was about equal to that passed during the day, but the **amount excreted was always greatest 2 or 3 hours after meals.** When fasting, the quantity sank to about half of the usual amount. **Ingestion of tyrosin increased the quantity.** Phenylacetic acid and phenylpropionic acid had no notable effect. The increase a short time after taking food seems to indicate that the homogentisic acid was produced from tyrosin, through the action of bacteria in the upper part of the intestine. The author, however, considers that this is probably not the case, and believes **that it is produced in the organism.** He thinks that it is **probably a normal product of intermediary metabolism.** Certain persons lose the power of oxidizing this substance; hence, his theory concerning the disturbance is one entirely analogous to the general belief regarding the origin of diabetes.

E. Meyer[2] reports a case of **marked alkaptonuria in a child 18**

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[1] Deut. Arch. f. klin. Med., Bd. LXXI, H. 1.

[2] Deut. Arch. f. klin. Med., Bd. LXXX, Hefte 5 u. 6.

months old. The general health was entirely undisturbed. He found, for a child, extremely large amounts of homogentisic acid, the quantity reaching 3.5 grams per day. The amount was greatest after the use of albuminous food. The acidity of the urine varied directly with the amount of homogentisic acid. The conjugate sulfates were not altered, the ammonia was very slightly changed, the latter fact indicating that the excessive production of acids is not great enough to cause any disturbance in the organism.

Oxaluria.—N. Stradomsky[1] discusses **the production of oxalic acid in the human organism.** This acid is a constant and normal component of the human urine, even when the food contains very little preformed oxalic acid. About 0.015 gram are excreted in 24 hours by an adult. Ordinary mixed diet causes the most marked excretion; a diet composed largely of meats produces the next largest excretion; next to this stands a fatty diet; and **least of all, an ordinary carbohydrate diet.** The increase after the use of meats is due to the colloid substances in the meat, and perhaps to the creatin, but not to the albumins or nucleoalbumins. Diet containing preformed oxalic acid causes increased oxalic acid excretion; viz., an alimentary oxaluria. **Oxalic acid is, however, produced in the human organism, probably chiefly from colloids,** and perhaps from creatin. When oxalic acid was given by the mouth, 35.3 % was found again in the feces and urine; the remainder was undoubtedly destroyed in the intestinal tract or in the organism, and **probably a portion of it was actually destroyed by the tissues.**

H. Salomon and L. Mohr[2] found that when a diet free from oxalic acid was used, small quantities (2 mg. to 5 mg.) of oxalic acid could be found in the 24 hours' urine. This was attributed to the production of oxalic acid in the tissues. The authors believe that **the organism undoubtedly oxidizes oxalic acid,** since only a small quantity of that administered appeared in the urine. They could not discover any direct relation between the nitrogen and the oxalic acid, but the use of gelatin and of tissues rich in connective tissue caused a marked increase. The use of nucleins produced doubtful results. They think, however, that oxalic acid **may be produced from uric acid.** Oxaluria can no longer be considered to be a special disease; it is a condition which is observed in a number of actual diseases. The authors found the oxalic acid increased in a neurasthenic, and attribute the increase in this case to hyperacidity of the gastric juice. It was not increased in certain cases that they examined in which it is commonly supposed to be increased; viz., diabetes, icterus, nephritis, gout, pneumonia, and leukemia.

A. Cipolinna,[3] in a study of oxalic acid in animal organs, has found that **the thymus of calves contains the greatest quantity;** after this, the spleen. Human organs contain a practically constant amount, the greatest quantity being found in the spleen; the next, in the kidney, the lungs, the liver, the muscles, and the brain. The blood contains only traces. The whole human body contains, he believes, only about 0.29 gram of oxalic acid. **Spinach contains more than any other food;** then sorrel, Brussels sprouts, beans, melons, cabbage, cauliflower, and carrots, in the order in which they are mentioned. Oxalic acid is probably produced in the organism as an oxidation product of uric acid, and is probably produced chiefly in the spleen; very likely, also in the liver and muscles.

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[1] Virchow's Archiv, Bd. CLXIII, Heft 3.
[2] Deut. Arch. f. klin. Med., Bd. LXXX, Hefte 3 u. 4.
[3] Clin. med. italiana, No. 10, 1901.

G. Pierallini[1] discusses alimentary oxaluria. He investigated the amount of oxalic acid in the urine with normal diet, and after administering foods containing large amounts of oxalates, or actually giving oxalic acid or calcium oxalate. The use of the acid itself, or of foods containing it, always caused an increase in the excretion. **Even calcium oxalate causes the excretion of an excess.** The smallest increase was seen after the use of vegetable substances which contain it, such as spinach and tea. The author decides that the soluble and insoluble salts of oxalic acid—the latter to only a slight degree—are absorbed, and that **the quantities in foods are sufficient to produce an increase in the excretion.**

G. Klemperer and F. Tritschler[2] contribute a study as to **the source and solubility of the oxalic acid of the urine.** They first state that of 16 cases of kidney calculi which had been passed spontaneously by the patients, they found that 3 were composed of calcium oxalate alone; 2 others contained that substance with others. A similarly high percentage of oxalate calculi has been found by a number of other observers. The authors found that oxalic acid, when given with the food, disappears to a large extent. A small portion of it was found in the urine, and an even smaller portion in the feces. Probably the intestinal bacteria break it up. They found that **after directly adding it to the feces a very large portion vanished. The blood, also, can apparently destroy oxalic acid,** and the authors believe that their experiments show that the blood can produce oxalic acid from uric acid, although the amounts produced were so extremely small as to make the conclusion subject to question. Subcutaneous injection of sodium oxalate in a dog was followed by increase in the oxalates of the urine. Calcium oxalate did not notably increase the excretion. The subcutaneous injection of sodium oxalate in a man, however, caused a decided increase of the excretion. They thought that uric acid, as previously suggested, might cause an increase; the ingestion of large amounts of uric acid, however, caused no notable increase in the excretion; but **glycocoll did increase the excretion decidedly.** Probably, therefore, gelatins would increase the excretion. Creatinin would also, because it is a substitution product of glycocoll, be likely to have the same effect; and it was found that, when used in quantities of about 2 grams, it did decidedly increase the excretion of oxalates. They decided that the oxalic acid of the urine comes chiefly from the vegetable food; that probably about one-tenth of that ingested is absorbed and excreted as oxalates in the urine; that the part that is not absorbed is destroyed by the intestinal bacteria; that, however, a **certain portion of the oxalates of the urine comes from the animal food, from the creatin, and from the foods which provide glycocoll;** and that a portion of the oxalic acid of the urine is perhaps derived from the reabsorbed portions of the bile, since glycocholic acid furnishes glycocoll in the intestine. The authors then discuss their studies of the solubility of the oxalates in the urine, and the effects of various foods and mineral substances, and decide that the **solubility of the oxalates depends upon their relative and absolute amount.** The conditions are best for solubility when the amount of oxalic acid is from 1 to 1.5 mg. to 100 cc. of water. **A marked acid reaction of the urine increases the solubility, as does also a high amount of magnesia salts with a low amount of calcium salts.** A proportion of calcium to magnesia of 1 : 0.8 up to 1 : 1.2 is best, and the amount of magnesia should be, if possible, as much as 20 mg. to 100 cc. of urine. All these **conditions are most satisfactorily approached when the food contains a large amount of meat, pastry, and leguminous vegetables,** and the patient takes large quantities of fluid. Milk, eggs, and other vegetables than the leguminous variety, should be excluded. The magnesia content of the urine can be increased by a prolonged use of small doses of magnesia salts; and in this way the solubility of the oxalates can be increased.

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[1] Virchow's Archiv, Bd. CLLX, Heft 1.

[2] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 3 u. 4.

J. A. Wesner[1] reports some investigations of the **relation between indicanuria and oxaluria,** and of their relation to gastrointestinal fermentation. He says that oxalate crystals in the urine commonly indicate gastrointestinal fermentation; that the presence of oxalate crystals does not necessarily indicate a high acid-percentage; that indicanuria is often—but not necessarily—associated with oxaluria; that hyperacidity with a meat diet tends to increase intestinal putrefaction; and that in some gastrointestinal disturbances due to an excess of hydrochloric or fatty acid, associated with fermentation, indican and oxalic acid are both increased. The symptoms of the so-called oxalic-acid diathesis and of the condition associated with indicanuria are not due to the oxalic acid or to the indol, but to other products formed in the process of fermentation. **Oxaluria and indicanuria are, therefore, indicative only of putrefaction.**

Indicanuria.—E. Reali[2] notes that indoxyl appears in the urine combined with glycuronic acid, as well as with sulfuric acid. The indoxyl may be freed from the glycuronic acid by boiling with vegetable acids, such as lactic acid. If, at the same time, barium is added to the urine, the glycuronic acid will go down with the barium precipitate, and may be isolated in this way. The presence of **indoxyl-glycuronic acid** in the urine is, the author believes, an **evidence of metabolic disturbances, and is not attributable to gastrointestinal disorder.** He considers that it **occurs frequently in arthritic diseases.** Since glycuronic acid is a product of the metabolism of carbohydrates, the variations in its amount in the urine are coincident with variations in the oxidative processes of the organism. [A statement which cannot be brought into accord with the teaching of Emil Fischer.] Reali considers that **uroerythrinuria with excessive indicanuria indicates anatomic changes in the liver.**

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[1] Chicago Med. Recorder, July, 1901. [2] Wien. med. Woch., Nos. 34 to 36, 1901.

H. Strauss[1] reports a new clinical method for the **quantitative determination of the indican of the urine.** He first makes a permanent **test-solution of indigotin,** containing 1 mg. of this substance in 100 cc. of chloroform. The test is then carried out by precipitating 20 cc. of urine with 5 cc. of 20 % sugar of lead. Of this filtrate, 10 cc. is placed in a separatory funnel [he uses the graduated funnel which he has previously described in the estimation of the lactic acid in the stomach-contents]. He adds 10 cc. of Obermayer's reagent, and then 5 cc. of chloroform. The funnel is inverted several times, and then allowed to stand for 2 or 3 minutes. The chloroform is then run off, and the extraction is repeated until the chloroform is colorless. The total amount of chloroform-extract is noted; 2 cc. of it is taken, and diluted until the color of it is the same as that of the test solution. The amount of indigo blue is then reckoned by the formula $x \frac{y}{2}$; x meaning the total chloroform extract in cubic centimeters; and y, the amount of chloroform in the final test, which is compared with the indigotin solution. The author has found values varying from below 5 up to as much as 168. The results were considered by Salkowski to be **sufficiently accurate for clinical purposes.** Reckoned in milligrams, according to the test-solution, the values varied from 2 to 4 mg. in normal persons, up to over 60 in urines rich in indican,—this amount being the 24 hours' quantity. The method can be carried out in about 20 minutes.

C. Rössler[2] has made some investigations concerning the **nature of the substance which causes the peculiar brownish coloration** in many instances when one is testing for indican in the urine. It is particularly likely to appear when Obermayer's test is used; but is also frequently observed after using mere fuming HCl, and shaking with amyl alcohol. The pigment is always extracted by amyl alcohol. Its nature had not previously been definitely determined, but Rössler found that it did not give the reactions for indigo red or urorosein, and hence he believed that it must be skatol red. He therefore isolated the pigment after having given the patient considerable quantities of skatol, and found that he obtained with the pigment very **distinct reactions for skatol.** The method of testing which he recommends is as follows: 10 cc. of fresh urine is added to the same amount of fuming HCl; after 5 minutes 5 cc. of amyl alcohol is added, and the whole is well shaken. In some cases there will be at once an evident brownish-red coloration of the amyl alcohol. If the color is mixed with blue, or if the urine is already old, one should first precipitate with lead subacetate, filter, and then add fuming HCl, and also chloroform to extract the indigo blue and indigo red. The supernatant fluid should then be shaken with amyl alcohol. The clinical importance of a positive result of the test is still to be determined.

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[1] Deut. med. Woch., April 17, 1902. [2] Centralbl. f. innere Med., Aug. 31, 1901.

MacPhedran and Goldie[1] report a **case of indigouria** in a man of 24 who presented no recognizable organic changes. The urine contained neither albumin nor sugar, and microscopically showed nothing except amorphous masses of indigo-colored pigment and blue coloration of the normal formed elements of the urine. The urine itself had a cloudy, bluish appearance. The occurrence of indigouria in a person who has taken no medicine, and is not the subject of any gastrointestinal disturbance or other acute disease, is very unusual.

Bacteriuria.—B. Goldberg[2] reports a case of postgonorrheal vesical bacteriuria, due to **Bacterium lactis aerogenes**—the second case ever reported as due to this organism. The man had had gonorrheal urethritis for about 9 months, but had become practically well. Suddenly the urine appeared cloudy, and at the end of micturition a white substance was passed, which proved to be composed almost entirely of short, rod-like organisms, which further investigation showed to be Bacterium lactis aerogenes. The patient **improved rapidly under the use of urotropin and intravesical injections of silver nitrate.** The urethritis persisted to some extent at the last report, but was much improved.

A. Wanner[3] has made an elaborate study of the usefulness of **various disinfectants of the urine,** and reaches the conclusion that **urotropin has decidedly the most marked action,** bacterial growth being practically entirely interfered with, the colon bacterium alone showing only a very greatly reduced activity. **The growth of the bacteria is at first interfered with; subsequently the bacteria are killed.** Tannopin and tannoform had no effect, nor did boric acid, guaiacol, or creasote. Benzoic acid reduced the growth of the streptococcus when used in large amounts, did not affect other bacteria, and had no influence at all in small doses. Salol interfered with the growth of the streptococcus, but had little influence upon other organisms.

Hematuria.—L. G. Guthrie[4] discusses **idiopathic or congenital hereditary and family hematuria.** He states that he knows of 12 cases of idiopathic hematuria, and that 8 of these are children of the same parents or first cousins. The mothers of these children, sisters, are themselves subject to hematuria, and these women had two brothers who had hematuria.

Goldsmid[5] reports 2 cases in which **hematuria followed the use of urotropin.** In both cases it disappeared after the use of the drug was discontinued. The doses employed were 7½ and 9 grains t. i. d.

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[1] Brit. Med. Jour., Oct. 12, 1901.
[2] Centralbl. f. innere Med., Mar. 29, 1902.
[3] Centralbl. f. Härn- u. Sexualorgane, vol. XII, No. 11.
[4] Lancet, May 31, 1902.
[5] Australas. Med. Gaz., Sept. 20, 1901.

DISEASES OF THE KIDNEYS.

Nephritis.—Ricci[1] has made a study of **metabolism in chronic parenchymatous nephritis,** and decides that **when the nitrogen balance is positive it is generally to be considered as a desirable condition, and not as the result of imperfect excretion.** It usually indicates that the nitrogen retained is being built up into tissue, and is replacing the more or less constant tissue loss that tends to occur in nephritis. It is, therefore, desirable to attempt to produce a positive nitrogen balance in the disease, by the use of a free, readily assimilable, albuminous diet, excluding, of course, those cases in which there is marked digestive disturbance or a decided tendency to uremia. The author **does not consider a milk diet to be essential or the most valuable.** He believes that an alimentary albuminuria may occur.

J. Marischler[2] contributes an experimental investigation concerning the **influence of sodium chlorid upon excretion through the diseased kidney.** He investigated the conditions in 7 cases throughout several periods, first giving a diet in which the nitrogen, chlorin, phosphorus, and calcium were known, and determining the excretion of these substances in the urine and feces; in the next period continuing the same diet, but adding 6 grams of sodium chlorid daily; and in the third omitting the sodium chlorid and determining its after-effect. The general conclusions which he reaches are: that in parenchymatous nephritis the kidney allows of the passage of sodium chlorid, even when diuresis is decreased, while if any retention of the chlorids occurs, it is attributable to retention of water; the **diuresis after the use of sodium chlorid is likely to be decidedly increased in parenchymatous nephritis** if the amount of water given is much increased; if the amount of water given is not increased, the excretion of water is not likely to be influenced; **in interstitial nephritis the diuresis is likely to be greatly increased,** and there may be an actual loss of water from the tissues. The patients showed a tendency to retain nitrogen in the period in which sodium chlorid was not given, but this was not always the case. In the sodium chlorid period the nitrogen was increased.

H. Strauss[3] has made a study of the **molecular concentration of the sweat in a series of subjects who had nephritis** and in others who did not have this disease. Very variable results were obtained. The concentration was often high in nephritis, but, as a general rule, was less than that of the blood-serum. The sodium chlorid content was also very variable, and was usually not more than 0.5 %. If the concentration due to sodium chlorid were subtracted from the total, the chlorin-free molecules were always found less than in the blood-serum. **The secretion of sweat, therefore, carries off from the body relatively more water than material in solution.**

E. Ekgren[4] has investigated in 3 cases the **influence of massage upon the albuminuria of nephritis.** He found that the amount of urine and the specific gravity were little influenced by the procedure; but that, whether massage and resistance movements were confined to the upper extremities or were given generally over the upper and lower extremities and the trunk, the **albuminuria was regularly increased.** He believes that these results indicate that massage and resistance movements should not be given unless it has previously been learned whether the patient has or has not albuminuria; also, that the use of massage for the removal of the edema of nephritis is a procedure associated with danger; and, if used at all, it should be controlled by frequent examination of the urine.

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[1] Il Policlinico, 1901, Fasc. IX.

[2] Arch. f. Verdauungs-krankheit., Bd. VII, Hefte 4 u. 5.

[3] Fortschritte der Med., 1901, No. 21.

[4] Deut. med. Woch., Feb. 27, 1902.

J. J. Pringle,[1] in a discussion of **the skin eruptions which occur in Bright's disease,** refers to the frequency of pruritus. Urticaria, contrary to the statements that are often made, is rare. Eczema has not been shown to be frequently due to nephritis. The most characteristic and interesting skin affection is the **uremic papular erythema;** it is, however, rare. A recent collection of cases showed only 46 recorded. The appearance of this eruption is ordinarily **a sign of very great gravity.** It is most common in interstitial nephritis. General bullous eruptions in rare instances occur in Bright's disease, but probably have no direct relation to the nephritis. General exfoliative dermatitis may occasionally be attributed to chronic nephritis. There is nothing definitely known concerning the actual causation of the skin eruptions.

E. B. Bostock and J. G. Emanuel[2] report a case of albuminuria of obscure origin occurring in a case of thoracic aneurysm. Upon postmortem examination the albuminuria proved to be due to lardaceous disease of the kidney, but the **cause of the lardaceous disease was obscure.** Amyloid changes were very widespread—much more so than is common as a result of uncomplicated syphilis; and yet no other cause could be found, and the patient was probably syphilitic. It was notable that while syphilis generally produces diffuse amyloid spleen, and tuberculosis sago spleen, in this case sago spleen was present. The case demonstrates that **lardaceous disease of the kidneys and aneurysm, either alone or combined, are not sufficient to produce hypertrophy of the left ventricle;** since it was absent in this instance. The patient had exhibited pronounced rigors occasionally in the course of his disease. These were apparently due to the accumulation of a mucopurulent secretion in the bronchial tubes, as a result of compression by the aneurysm.

W. Vysin[3] discusses the **relations of nephritis to hydremia and dropsy,** and decides that in hydremia the dilution of the blood-serum may not be proportionate to the dilution of the total blood, the latter being relatively more marked. The **amount of albumin excreted has no influence upon the specific gravity of the blood,** nor upon the numbers of the red blood-cells and the hemoglobin content. The specific gravity is, in most cases, reduced, both in persons with dropsy and in those without. The cause of this is some unknown factor, which causes the retention or the imperfect excretion of fluid. He considers that the **dropsy is produced by changes in the blood-pressure.** The grade of the dropsy is not always in direct relation with the specific gravity of the blood. The reason for the latter fact is found in variations in transpiration through the skin. There are no definite relations between hydremia and dropsy.

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[1] Practitioner, Nov., 1901. [2] Birmingham Med. Rev., July, 1901.
[3] Wien. med. Woch., Nos. 38 and 41, 1901.

Uremia.—J. R. Bradford,[1] in a **general discussion of uremia,** insists that it has been **impossible to correlate uremia definitely with the existence of high arterial tension.** He refers to his own work, which, he believes, has shown that experimental lesions involving complete double nephrectomy lead to the production of latent uremia, but never to that of acute or chronic uremia; in other words, they produce symptoms similar to those observed in calculus obstruction or in sudden obstruction of the renal arteries. As to the toxicity of the urine, he has found that its toxic action does not disappear in uremia, and may be, at any rate, quite as high as that of normal urine. **The great argument against uremia being due to the retention of toxic substances normally excreted in the urine** is that the picture of calculus suppression is that of latent and not of acute uremia; also, acute uremia is not infrequent when there is little or no suppression. The author insists upon the **importance of the wasting that occurs in Bright's disease as an indication of a disorder of metabolism,** and refers to the fact that he found that the removal of a large quantity of kidney-substance was followed by extreme wasting, although the animals had a fair appetite and did not suffer from any digestive disturbance. The kidney seems, therefore, to have an influence upon nitrogenous metabolism; but Bradford insists that the **lack of an internal secretion cannot be the explanation of uremia,** because the phenomena observed after double nephrectomy are identical with those seen after bilateral ligation of the ureters, and because complete obstruction of the renal arteries in the human subject is not followed by acute but by latent uremia. He also believes that there is **no satisfactory evidence of the existence of an internal secretion of the kidneys.** The only well-established facts about uremia he considers to be: (1) That the retention of the normal constituents of the urine is not sufficient to produce uremia; (2) that in uremia there is extensive destruction of the proteid tissues of the body; (3) that while in many cases of uremia in the final stages there is more or less complete suppression of the urinary secretion, this is not invariable or essential, and an abundant secretion may be present. In the symptomatology of uremia the author states that **dyspnea is the most constant symptom,** and very frequently the initial one. Complete suppression of urine is rare in acute uremia. While albumin may be absent from the urine, this is of marked rarity. Acute uremia is more common in chronic renal disease than in acute nephritis; latent uremia may occur in chronic renal disease, particularly in cystic kidney. Acute uremia Bradford believes to be more common in contracted white kidney, though not uncommon in the large white kidney. Ordinarily the eclamptic form of uremia is not so fatal as the comatose variety. The presence of **marked dyspnea is a very unfavorable sign.** Uremic rashes are usually believed to be of very evil import, but such cases are not always fatal. The **use of renal extract has not, he thinks, shown itself to be of any value.**

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[1] Practitioner, Nov., 1901.

Strubell[1] **believes that he has demonstrated that the experimental uremia produced in animals by extirpation of the kidneys is not different from the uremia observed in man.** He thinks, therefore, that results obtained in experimenting with dogs may be considered to be useful in studying the condition in human beings. He especially discusses the diet, and insists that a purely carbohydrate or, at least, a purely vegetable diet is best in such cases. He says that this diet may be considered to be the direct opposite of a diabetes diet, and that everything that is excluded from the diet of the diabetic should be allowed—alcohol of all kinds, however, being forbidden.

A. Bickel[2] discusses the **relation between the changes in the freezing-point of the blood and nervous disturbances,** particularly referring to the relation that this question bears to uremia. It has been strongly indicated by the work of a number of investigators that a pathologic increase in the amount of sodium chlorid in the circulation results in specific poisonous effects upon the nervous system, and that these effects are not directly due to increase in the molecular concentration of the blood, and consequently in the osmotic pressure. Bickel reports his results from the **injection of sodium chlorid solution into various animals.** He found that in rabbits, for instance, the results are slowing of the pulse, and various nervous symptoms of the irritative and paralytic types. The reflexes become increased, and finally there are tonic and clonic convulsions, which appear at intervals. Preceding these there are fine tremors, also general tremors of the extremities, and at times nystagmus. **The animals fall into deep coma, and remain so until death.** The symptoms differ to a certain extent in different animals. In rabbits if the injection is undertaken very slowly, convulsions may readily be postponed until the terminal stage, and sometimes they do not occur at all. With dogs, on the contrary, it is practically impossible to avoid the occurrence of convulsions just before death. These results are interesting in their relation to uremia. Lindemann has claimed that uremic convulsions are due to increased molecular concentration of the blood, and increased osmotic pressure. In order to disprove this statement Bickel refers to the results of a number of other workers who have found that very low freezing-points may be observed without any signs of convulsions, and, on the other hand, convulsions may occur when the freezing-point is not very greatly depressed. He also adds, as further testimony of the truth of his views, the records of his results with a number of animals. As an example of these it may be mentioned that he repeatedly found the freezing-point at about —0.90° C. after injections of sodium chlorid, given slowly, and yet there were no signs of convulsions, and convulsions appeared only when the freezing-point had been depressed still lower. These results are also confirmed by the work of Bickel and Schreiber with grape-sugar injections. With such injections they were able to reduce the freezing-point as low as —1.00° C. without causing any signs of convulsions. In general, it may be stated that an **injection of salt solution causes irritative nervous symptoms only when the concentration of the blood has become much greater than is observed even after extirpation of the kidneys.** He believes, therefore, that the **increase in the molecular concentration of the blood in uremia is only an evidence of retention** in the blood of an abnormal amount of substances which should be excreted, but this increased concentration alone is **not sufficient to explain the symptoms of uremia.**

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[1] Wien. klin. Woch., No. 29, 1901.

[2] Deut. med. Woch., Sept. 5, 1901.

E. Schreiber and J. Hagenberg[1] report a series of investigations which were undertaken for the purpose of determining the **nature of the influence of bleeding and salt infusion in uremia.** They compared the blood of 2 uremic human subjects and that of 2 dogs that were made uremic by bilateral nephrectomy, both before the use of salt infusion and half an hour later. **Neither bleeding nor salt solution infusion had any influence upon the freezing-point of the blood, but its viscosity** (determined by Jacoby's method) **had in each instance decreased**—although not notably influenced previously by the uremia itself. The viscosity of the serum had changed similarly, although less markedly. This, the authors believe, should relieve the strain upon the heart and thus improve the symptoms. In a fifth investigation, however, they found that a uremic patient did not exhibit the same blood-changes; indeed, after the second bleeding which they undertook, they found a marked increase in the reduction of the freezing-point and a decided increase in the viscosity of the total blood, and of the serum. It is possible that the increase in the reduction of the freezing-point was due to taking up toxic substances from the tissues after the bleeding, and that the increase in the viscosity was due to the marked improvement in the condition of the blood, which had been extremely anemic, but increased from 1,568,000 to 3,356,000 red cells.

W. H. Broadbent,[2] in a general discussion of **renal disease and the circulation,** insists that the cardiovascular changes play an important part in the production of the phenomena which are usually said to be toxic. He **believes that uremic convulsions are**—to a considerable extent, at any rate—**due to circulatory changes,** chiefly stasis of the cortical cerebral circulation. Among other reasons for this, he states that he has seen severe convulsions occur, with very high arterial tension, in the absence of renal disease; and refers to the case of a medical man who had had violent convulsions 20 years before. There was extremely high arterial tension. Bleeding reduced the tension, and the man rapidly recovered, and has always remained well since. **Cheyne-**

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[1] Centralbl. f. Stoffwechsel u. Verdauungs-krankheit., No. 11, 1901.

[2] Practitioner, Nov., 1901.

Stokes respiration he also considers to be due to the high arterial tension. The therapeutic indications are free venesection in uremic convulsions, and the use of a calomel purge. The venesection should be repeated, if necessary. He states that the **best general means of reducing a high arterial tension is the administration of mercurial aperients.**

Laquer[1] contributes a report of 2 cases of uremia in which he **observed a curious reaction of the blood-serum when used with the blood of another species** (rabbit). The same fact was observed before by Neisser and Doering. The authors last mentioned stated that if 0.1 cc. of the serum of their uremic patient was added to 1 cc. of rabbit serum it gave the usual reaction, but when inactive serum from the patient (serum that had been heated to 55° C.) was added it **interfered with the reaction due to the active serum,** and the mixture of active serum with rabbit's blood then remained unchanged. An exactly similar condition was seen in the two cases which Laquer reports. He has examined a large number of specimens of blood-serum from other patients, and has **never seen this reaction under other circumstances.** That it was due to uremia seemed to be demonstrated by the fact that in one of these patients **the reaction was not present before the onset of the uremia, but was seen after the development of uremia. A difficulty in using this reaction in the study of uremia** is found in the fact that the activity of reaction of unheated serum may be greatly reduced without the onset of uremia; while this is not the curious reaction of which Laquer writes, it interferes with the demonstration of the presence of such a reaction. He notes that in 2 cases of uremia the blood-pressure was very high: in one case 180 to 185; in the other case 215 to 220, as estimated by the Gärtner tonometer. He found a **decided reduction of the blood-pressure after the use of wet packs.** He believes that the tonometer is very useful in prognosis and diagnosis in uremia, and that it may indicate the advisability of certain therapeutic measures.

Treatment of Nephritis.—Kauffmann and Mohr[2] contribute some experimental results concerning **the use of various kinds of meat in diseases of the kidney,** particularly directing their attention to the question of the relative harmfulness of light and dark meat. They describe 7 experiments of their own, and also refer to the results of v. Noorden and Ritter in two other cases previously reported. They reach the conclusion that there are such marked variations in the excretion in cases of renal disease that it is difficult to decide upon the exact importance of any results obtained in a study of nitrogen-excretion in kidney disease with various forms of diet; but, taking it all in all, there is **no evidence that light meat is associated with any better nitrogen excretion than is dark meat, and the excretion on milk diet is not notably better,** although it seems slightly better. The authors admit that the brevity of the periods through which the cases were studied makes it possible that their conclusions are erroneous; but the cases could not be studied longer, and, taking their results in connection with the results of clinical observation and in connection with the observations of Offer and Rosenquist, they decide that we are perfectly justified in believing that the general dogma that dark meat is more harmful than light meat in kidney disease is erroneous, and that various forms of meat are suitable in such cases purely in proportion to their digestibility. These conclusions they consider valid in chronic and subchronic nephritis only. It is, at best, doubtful whether a diet containing meat should be allowed at all in acute nephritis.

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[1] Deut. med. Woch., Oct. 24, 1901.

[2] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 5 u. 6.

Elliot[1] discusses the **employment of diuretics in different conditions of the kidney.** In functional disturbances in which the difficulty is chiefly with excessive concentration and excessive acidity of the urine, diluents and salines are best; and in actual nephritis pure saline treatment is likely to be most satisfactory. In severe cases subcutaneous salt infusions are usually important. In chronic nephritis the diuretics which act chiefly upon the heart and vessels are usually most satisfactory. Diuretics have practically no influence upon dropsy of purely renal origin.

Cystic Kidney.—W. Osler[2] reports 2 cases of bilateral cystic kidney and refers to the following points as being especially important in the diagnosis: (1) The presence of bilateral tumors in the flanks; (2) the cardiovascular changes of interstitial nephritis; (3) the condition of the urine found in advanced interstitial nephritis; (4) hematuria. The recognition of bilateral cystic kidney is extremely **important in cases in which nephrectomy is contemplated.**

Sarcoma of the Kidney.—A. Rothschild[3] reports the case of a man of 59 years who was seen chiefly because of hematuria. His **urine contained a peculiar, worm-like body,** with a smooth, glistening surface, having the shape of the ureter. It was suspected that it might be a portion of a tumor. Microscopic examination, however, showed that it was **practically homogeneous and contained no cells.** Chemical examination and staining showed that it was not fibrin. It was impossible to make a definite diagnosis in the case at that time, but the man subsequently appeared with the evidences of cachexia and with an easily palpated tumor in the region of the left kidney. That organ was removed, and there was found a **giant-cell sarcoma,** involving the upper two-thirds of the kidney. Part of the tumor was of a grayish-red appearance, while the remainder was **of the same peculiar, homogeneous structure as the body found in the urine.** Portions of the latter substance projected into the pelvis, and small fragments were found free in the pelvis. At the time of operation one of these was in the ureter. This substance showed the same staining and chemical characteristics as did the body found in the urine, and it was, therefore, decided that the latter was derived from the tumor. Rothschild insists upon the fact that it is not possible to exclude tumor if the bodies found in the urine, although suspected to be portions of tumor, prove to be of other nature—a somewhat self-evident fact, but one which is not likely to be appreciated under such circumstances. At the end of the paper the note is made that the examination of the substance had also shown that it was not mucin. A review is then given of the 7 other cases of coagulum-like bodies in the urine that have been reported.

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[1] Brit. Med. Jour., Aug. 10, 1901.

[2] Amer. Med., Mar. 22, 1902.

[3] Deut. med. Woch., Dec. 12, 1901.

PARASITES.

J. Chalmers Da Costa and R. S. Dorsett[1] report an **important case of sporadic trichinosis,** which was remarkable for the fact that there was **no fever, and upon repeated blood-examination there was no eosinophilia.** The diagnosis was finally made by excising a portion of the muscle and discovering the parasites therein. The authors consider the case to be evidence that the diagnosis of trichinosis can at times be made only by removing a piece of muscle and examining this, even when other evidences of the disease are absent. R. C. Rosenberger discusses the pathologic findings and the literature concerning trichinosis.

W. J. Calvert[2] reports **4 cases of filariasis,** 2 of which were known not to have trichinosis. **All of them showed decided eosinophilia.** In cases of long standing an increase in eosinophiles is not found. The author states that in the early stages of filariasis one may expect to find leukocytosis with increase in the eosinophiles; in the latter stages these conditions will probably not be found. In one case the eosinophiles went above 20 %.

G. L. Gulland,[3] in discussing the condition of the blood in filariasis, reports a case in which he made repeated differential counts and found from **3 % to 12 % of eosinophiles.** The eosinophiles were **largely increased in the middle of the night, as compared with the condition during the day.**

Low,[4] in some studies on **Filaria Demarquay,** states that this organism is readily discovered in the blood, and is easily distinguished from Filaria nocturna by its smaller size, more rapid movement, and less pronounced head characteristics. The evidences of disease which it produces in the human subject are much less marked than those following infection with Filaria nocturna. They are chiefly the **signs of local irritation or inflammation.** The intermediary host does not seem to be either the culex or the anopheles.

I. P. Lyon[5] gives a **review of echinococcus disease in North America, tabulating 241 cases.** He finds 60 % of the cases in males, these figures differing widely from those of most other authors. Of the persons affected, 91 % were foreign-born. The author insists upon the importance in prophylaxis of avoiding close contact with dogs, and also emphasizes the **necessity for proper sanitary regulations**

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[1] Am. Jour. Med. Sci., Dec., 1901.
[2] Johns Hopkins Hosp. Bull., June, 1902.
[3] Brit. Med. Jour., April 5, 1902.
[4] Brit. Med. Jour., Jan. 25, 1902.
[5] Am. Jour. Med. Sci., Jan., 1902.

at slaughter-houses, which shall demand that the offal of slaughtered animals shall not be exposed where dogs can reach it.

F. Billings and J. L. Miller[1] report that in the urine of 2 patients they discovered the presence of **Anguillula aceti.** The parasite was at first thought to be Strongyloides intestinalis, which it closely resembles.

S. E. Henschen[2] discusses the **pathogenetic importance of Balantidium coli.** He reports 2 cases, in both of which the symptoms ran a course which varied from time to time in direct relation to the number of parasites present, in both of which the patients recovered after the entire removal of the parasites, and in both of which the diarrhea had no other apparent cause. He has, in all, seen 8 cases. In 5 recovery occurred practically at once after the removal of the parasites; in one, improvement occurred, but the patient disappeared from treatment; in another the patient recovered about a month after the parasites were removed; and in the remaining case the diarrhea did not disappear, although the parasites were removed. In the latter case, however, there had been a chronic colitis for over 2 years, and this probably caused the continued disturbance. Henschen directs attention to the fact that he has previously been repeatedly quoted as believing that these parasites are not of pathogenetic importance. This is a mistake due to the fact that about 30 years ago he published a paper, with Waldenström, in which such an opinion was expressed. This opinion was, however, the latter's, and not his own. He **believes that the parasites do actually produce many, and sometimes all, of the symptoms seen when they are present.** He has found **large enemas containing tannic acid and vinegar extremely valuable** in the treatment of these cases, recovery ensuing in most instances after short periods of treatment.

Erich Zabel[3] reports an interesting case of gastric carcinoma in which **Megastoma intestinale and other parasites were found in large numbers in the gastric contents.** The patient was operated upon, and a total extirpation of the stomach was carried out. The author gives an extensive discussion of the megastoma itself and of its importance from a pathogenetic and diagnostic point of view. He **does not believe that there is any indication that these parasites of themselves cause symptoms,** but he thinks that **their presence in persons who have no indication of intestinal disturbance points to disease of the upper intestine or of the stomach.** In most such cases there is achylia or hypochylia gastrica, and this is the direct cause of the infection, the normal HCl of the gastric contents killing the parasites. For the reason indicated, children who have had gastrointestinal disturbance and adults with achylia or hypochylia—the latter frequently including cases of phthisis—are the chief subjects of infection with these parasites. In the cases reported the infection probably occurred through the drinking-water. Examination of this water showed a fauna closely resembling that of the gastric contents. Besides the megastoma, the author found numerous forms of bacteria, with many spirilla, various forms of leptothrix, amebas, monads, cercomonads, trichomonads, and peculiar bodies which were probably of the class of infusoria.

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[1] Amer. Med., May 31, 1902. [2] Arch. f. Verdauungs-krankheit., Bd. VII, Heft 6.
[3] Arch. f. Verdauungs-krankheit., Bd. VII, Heft 6.

E. Becker[1] discusses the literature relating to **Trichocephalus dispar and the disorders caused by it,** and describes 2 cases which he has himself observed. He insists that this parasite may produce symptoms, which may be of the 3 following classes: (1) **Gastrointestinal symptoms,** with evidences of catarrh or ulceration of the colon; (2) **nervous symptoms,** which may become so severe as to resemble meningitis, and which in some cases seem to have produced death; (3) **symptoms referred to the blood**—viz., an anemia of the chlorotic type, but with more or less severe changes in the form and size of the red cells. In one of the cases reported the active symptoms disappeared entirely and almost at once, and the blood-condition began to improve rapidly, after the parasites had been killed with **benzine injections,** the latter being first used in the strength of a teaspoonful to a quart. This being found too strong, it was finally made 5 drops to a quart. After the first injection the parasites decreased, and the eggs soon vanished.

Sandwith[2] reports a note on the **entrance of ankylostoma embryos into the human body by means of the skin.** He rubbed water containing ankylostoma larvas into the skin of the thigh of a boy shortly before the boy's leg was amputated. The fluid dried rapidly, without leaving any redness behind. After the amputation the skin area was extirpated and hardened in alcohol. Sections were cut, and microscopic examination showed that **the larvas had penetrated into the hair follicles,** but not into the follicles of the glands. The author believes that they would have bored their way through the hair follicles into the subcutaneous tissue under ordinary circumstances, and that this indicates that these parasites may enter the body by means of the skin, particularly, however, through injuries of the hands or feet.

Pisenti and Mandolesi[3] note a **relation between ankylostoma and pellagra.** They found that in a number of cases of pellagra in which there was very severe anemia the eggs of the ankylostoma were present in the feces. They believe that these parasites are **probably related to the causation of the pellagra.**

H. B. Allyn,[4] in discussing the occurrence of **ankylostomiasis in the United States,** reports a case and refers to 8 others, the only ones of the affection in this country that he has been able to discover in the literature. The patient whose case is reported, after having previously resisted other treatment, **improved markedly under the use of thymol.** His red blood-cells had been reduced to 871,875, and his hemoglobin to 15%.

T. Claytor[5] gives a further report of a case of ankylostomiasis with autopsy studies. He believes that the condition is more frequent in America than is usually thought. The patient whose case is reported was from Virginia. For 3 years he had anemia, and, for a year, swelling of the face, abdomen, and feet. He had had no gastrointestinal symptoms. He was **treated with large doses of thymol**; and, in spite of the fact that this treatment was carried out for 2 months, **ankylostomas were persistently passed, up to the time of his death,** which occurred as the result of cerebral hemorrhage. The autopsy showed thickening of the small intestine, particularly the duodenum. Twenty worms were still present. The liver and spleen were notably enlarged; there was a large, subdural cerebral hemorrhage; and there were scattered areas of softening of the brain-substance.

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[1] Deut. med. Woch., June 26, 1902.
[2] Brit. Med. Jour., Sept. 14, 1901.
[3] Gaz. degli osped., No. 84, 1901.
[4] Amer. Med., July, 1902.
[5] Am. Jour. Med. Sci., Jan., 1902.

PEDIATRICS.

By LOUIS STARR, M.D., and ALFRED HAND, Jr., M.D.,

OF PHILADELPHIA.

MILK AND INFANT-FEEDING.

Moro[1] could find no bactericidal activities in human or cow's milk, but he found that the **blood-serum of breast-fed children** has much **greater bactericidal and hemolytic powers** than that of bottle-fed infants; he attributes this to the presence of alexins in human milk, and, although these cannot be demonstrated, he believes that they are taken from the blood-serum of the mother and combined with the casein through the action of the gland-cells; he advances these facts as an explanation of the importance of breast-feeding, and closes with Favorinus's remark, "A woman is the complete mother of her child only when she herself suckles it."

A. Schlossman,[2] director of an infant home in Dresden, discusses the **management of hospitals** for infants, emphasizing the need of having wet-nurses in such institutions, and shows his experience in lowering the infant mortality by this means. Flachs[3] describes the plan of getting the **wet-nurses** from the Maternity Hospital; each brings her own child with her, continuing to nurse it, and she also nurses and cares for one or more of the sick infants; in return for this she receives her maintenance free and also a cash premium, when the child intrusted to her makes a certain gain in weight. H. Peters[4] reports on the **amount of milk at a single meal** taken by breast-fed infants, giving an immense mass of data compiled in the same institution, the children being weighed before and after each nursing; this investigation showed that a child at the breast takes far more than the stomach is supposed to hold at any given age, without developing symptoms of gastrectasia.

Heubner[5] calls attention to the **increase in lime in the stools** of infants fed on cow's milk over that present when they were breast-fed, Bendix having found the ash when cow's milk was given to be 15 % and over, while human milk gives stools with only 3 % to 6 %; this would indicate that the change in the character of the movements, when a child is weaned, is not dependent on the badly digested casein of cow's milk, for the nitrogen in the stools is not increased.

Vildermann[6] advocates the use of **cow's milk as a galactagog** for nursing mothers, and illustrates by weight-charts the benefits resulting to the child when the mother drank 1.5 liters of milk daily; in 9 cases there were but 2 failures. Delobel[1] discusses the **indications for weaning** and the way to prepare the child for it, recommending gradual substitution beginning at the sixth month.

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[1] Jahrb. f. Kinderheilk., vol. LV, 1902.

[2] Arch. f. Kinderheilk., XXXIII.

[3] Ibid.

[4] Ibid.

[5] Rev. mens. des Mal. de l'Enf., Dec., 1901.

[6] Arch. de Méd. des Enf., July, 1901.

B. Bendix[2] brings up the importance of the individual **child himself as a factor in successful feeding;** this is illustrated by the not rare observations that some children will not thrive on any food that may be given, no matter how carefully the diet is regulated, while others will grow up in spite of most absurd and irrational feeding (*e. g.*, white bread dipped in weak coffee); as a further illustration the author gives the data in the development of a number of sets of twins, there being usually marked differences in weight between the members of each pair. C. De Lange[3] made bacteriologic studies of the number of **bacteria in the stools of healthy infants,** finding that breast-fed infants, as a rule, pass far fewer bacteria than do bottle-fed infants.

J. K. Friedjung[4] thinks that perhaps too much significance has been attached to the **low content of iron in human milk,** which is lower than the amount of iron eliminated by an infant, thus pointing to the probability that iron is deposited in the liver in excess before birth; as a result of his investigations, the author states that while a healthy woman's milk has a small amount of iron, it is nevertheless constant, and is therefore not without influence in the child's system; he could not find any lowering of this amount of iron as lactation progressed normally, but unhygienic conditions, sickness, etc., easily cause a decided lessening; the milk of some mothers apparently in good health whose infants were not thriving, was found to be poor in iron. The author analyzed 3 prepared milk foods for infants and found all of them to have a much smaller amount of iron even than woman's milk.

O. Cozzolino[5] investigated the action of various members of the **colon group of bacteria in souring milk** from cows, sheep, asses, and women; difficulties of technic interfered with positive results in connection with woman's milk, which, however, developed a lower degree of acidity than the others, although having nearly double the amount of milk-sugar; the author attributes this to some difference in the character of the casein, as milk from which the casein was removed did not reach so high a degree of acidity, variety for variety, as that which had been uncurdled. The author[6] has gone over the ground the second time, using milk that had been subjected to fractional sterilization for 8 days, the results agreeing with his earlier experiments.

The urgent need for **clean milk** with a low bacterial count is obtaining recognition more and more widely. The disadvantages of sterilization and pasteurization, while not great enough to overcome at all times the advantages, are nevertheless so real and important that the movement for clean milk is gaining force constantly. J. W. Wichmann[7] and A. Johanessen[1] come to the same conclusions. S. Monrad[2] refers to the generally accepted belief [?] that sterilization renders cow's milk as easily digestible as human milk, because in the test-tube it will coagulate in small curds, but he thinks that this is insufficient basis for such belief; he details 5 cases of atrophy getting worse on sterilized milk and recovering when raw milk was used. Gagnon,[3] as a result of studies of metabolism in 4 children fed for equal periods first at the breast, then on sterilized cow's milk, and then on sterilized human milk, concludes that **sterilization exerts no influence on the absorption of the proteids.** Spolverini[4] finds that the elimination of nitrogen and fat is increased with sterilized milk and digestive disturbances are more common than when raw milk is used. A. Hippius[5] has devised an apparatus for **pasteurizing milk at home,** the vessel having an air-jacket around the water-compartment and heat being applied by a lamp, the temperature of 65° C. being obtained for as long as desired; the author studied the effect of pasteurization with the apparatus on the chemical and biologic constitution of milk, and concludes that pasteurization for 2 hours is wholly satisfactory without there being any alteration in the taste or value of the milk.

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[1] Ibid. [2] Jahrb. f. Kinderheilk., vol. LIV, 1901. [3] Ibid.
[4] Arch. f. Kinderheilk., vol. XXXII. [5] Ibid. [6] Ibid, vol. XXXIII.
[7] Ref. in Jahrb. f. Kinderheilk., vol. LIV, 1901.

L. Concetti[6] insists on the **division of simple atrophy into a primary and a secondary form,** the first being rare and the other being common; he limits the term primary infantile atrophy to those infants who, because of an innate defect of the organism, are unable to use their nourishment, no matter how closely this conforms to the rules of physiology and hygiene; in order to gain properly it is necessary for a child not only to digest its food, but to assimilate it and convert it into the organized form of the living cell; for these purposes, in addition to the digestive ferments, there is needed an internal secretion of trophozymases; but just as the digestive juices are deficient in the newborn infant, so probably are the trophozymases, the child ingesting both of these in the mother's milk; the author's assistants, Luzzatti, Biolchini, and Spolverini, have shown that **woman's milk contains ferments** that are absent or present only in small amount in cow's or goat's milk, and that sterilization destroys whatever ferment is present; he therefore longs for clean milk such as is produced by the Walker-Gordon farms in the United States; the ferments present in human milk are not specific to the milk, but it has been found that certain of them can be made to appear in cow's milk by means of certain fodders; a greater field will be opened if the trophozymases can be made to appear in cow's milk in a similar manner; and if this is ever accomplished, the time may come when the artificial feeding of infants will give as good results as the natural. L. M. Spolverini[7] reports on the results of **feeding goats and cows germinating barley;** amylolytic and hydrating ferments were then found in the milk, which are not there normally, but which do exist in human milk.

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[1] Ibid.
[2] Ibid., vol. LV, 1902.
[3] Ibid.
[4] Ibid.
[5] Deut. med. Woch., July 18, 1901.
[6] Arch. de Méd. des Enf., Mar., 1902.
[7] Ibid., Dec., 1901.

E. Moro and F. Hamburger[1] describe a test for the **differentiation of human from cow's milk,** consisting in the addition of a few drops to hydrocele fluid; the former causes a coagulation, due probably to fibrinogen in the hydrocele fluid and fibrin-ferment in human milk, cow's milk having no such effect. J. Brodzinski[2] has studied Umikoff's reaction in human milk, and finds that it is most often present between the fourth and eighth months of lactation, it is not present all the time, and in some nurses it cannot be obtained; it is never present in cow's milk; the reaction consists in the development of a rose-violet color on the addition of 2.5 cc. of a 10% ammonia to 5 cc. of milk and warming to 60° C. R. Jemma and F. Figari,[3] as a result of experiments with dead cultures of Bacterium coli, found that they were not innocuous for animals, and they therefore draw the practical conclusion that milk should be sterilized as soon as possible after it is drawn.

L. Langstein[4] fed 25 infants successfully with **junket,** shaken in order to break the curd into small particles. J. Comby[5] reviews an article by Borde,[6] who advocates **cereal decoctions in infant-feeding** at 6 months of age, claiming that they are well borne, that they subdue and prevent gastroenteritis, rachitis, and chronic intestinal disturbances, all of which Comby disputes, and states that cereals should not be used until the tenth or twelfth month.

K. Oppenheimer[7] contributes a paper, copiously illustrated by weight-charts, of the **results he obtained in feeding infants with undiluted milk;** he divides the cases into 2 main groups with subdivisions; the first group comprised those under observation more than 4 weeks, and included: (I) 11 healthy children, all of whom showed a daily average gain of 25 gm., as compared with the Camerer-Biedert requirement of 19.7 gm.; (II) 36 moderately ill children, with a daily average gain of 23 gm., as compared with Camerer's requirements of 19.6 gm. for such children; (III) 11 atrophic children, 4 of whom throve, 7 not doing well, on full milk; the second main group, under observation less than 4 weeks, included 13 children who did not thrive and 20 who did, the latter showing a daily average gain of 25 gm. The author gives the following points for guidance in the successful use of the method: the start should be gradual, half milk and half water on the first day, and then on each succeeding day 100 cc. more of milk and 100 cc. less of water; the daily amount should not exceed 500 cc. in the first month, 750 cc. in the second, and 1000 cc. in the third and fourth months; there should not be more than 8 daily feedings in the first 3 weeks of life, 7 from the second to the fourth months, after which 5, or at the most 6, will be enough. The author states that there will be a little difficulty at first in accustoming the child to the relatively smaller amount at each feeding, but if too much is given, vomiting and diarrhea or constipation will result. [We do not see the reasonableness of using this method, for it does not suit every case, and its results are at least no better than can be obtained by feeding each child, even in dispensary practice, according to its needs, rather than according to one plan for all children.] Biedert[1] makes a similar criticism. M. Ostheimer[2] placed 3 children, aged 8, 11, and 13 months, on a diet of unboiled and undiluted milk, curing the chronic gastroenteritis, the rachitis, and the malnutrition.

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[1] Wien. klin. Woch., No. 5, 1901. [2] Ref. in Jahrb. f. Kinderheilk., vol. LV, p. 624.

[3] Clinica Moderna, 1901. [4] Jahrb. f. Kinderheilk., vol. LV, 1902.

[5] Arch. de Méd. des Enf., July, 1901.

[6] Gaz. hebd. des sci. méd. de Bord., April 18, 1901.

[7] Arch. f. Kinderheilk., vol. XXXI.

Romanoff[3] suggests a simple plan for obtaining a cow's milk mixture of the **same composition as human milk;** he dilutes milk with an equal amount of oatmeal-water containing 4 % of sugar; after sterilization the bottles are allowed to stand for several hours, when the lower half of each bottle is siphoned off and the upper half, containing about 3.5 % fat, is used for feeding.

A. Jacobi[4] elaborates his argument for the use of **cane-sugar in infant-feeding,** in preference to milk-sugar, as he doubts whether the latter when obtained from cow's milk is identical with that in woman's milk; and the milk-sugar of commerce is very often impure; the possible effects in the system also make cane-sugar preferable. [While we are in accord with such use, we are not dogmatic on all the points raised.]

C. W. Townsend[5] details an interesting result in the **feeding of an incubator baby,** whose weight at birth was 2 pounds, 12 ounces; this weight was doubled in 12 weeks, tripled in 4½ months, and increased more than 6 times by the end of the year, when the child weighed 17 pounds; although of such light weight, the child was born only 2 weeks before term, the probable cause being necrotic areas in the placenta associated with albuminuria on the part of the mother; the infant was fed on a home-modified milk, mixed according to the author's method [*vide* YEAR-BOOK for 1900], which consists in pouring off the top quarter of a bottle of milk after it has stood for 5 hours, viewing this as a 10 %-fat milk, and taking 1 ounce of this for every 0.5 % fat, 0.2 % sugar, and 0.2 % proteid desired in a 20-ounce mixture; in this case the infant was given the above percentages of fat and proteid with 4.2 % sugar (obtained by adding 1 even tablespoonful of sugar of milk) at the start, gradually raising the strength to fat 2.5 %, sugar 5 %, proteid 1 % by the seventh day; at 3 months the percentages were practically those of human milk—fat 4 %, sugar 5.6 %, albuminoids 1.6 %, made by taking top-milk 8 ounces, water 11 ounces, lime-water 1 ounce, sugar of milk 2 tablespoonfuls; after the eighth month cereals were used as diluents; at one time milk from Jersey cows, with a fat-percentage of 3.85, was used, but there followed an increase in the number of stools, with considerable wind, the digestion becoming normal with a return to a mixed milk of Holstein and Ayrshire cows.

T. de Mattos[6] quotes Heubner as saying that in the solution of the problem of infant-feeding what is needed is facts; he mentions the cost as among the disadvantages of some of the prepared foods, shows that it cannot be predicted with certainty which will agree, and that investigations of the feces (Monti, Biedert, and others) do not furnish accurate data; the American "individualizing" plan (Rotch) he terms experimenting on hypothetic or empiric bases with the proportions of breast-milk as a goal, but he quotes Eross, who found out of 1000 breast-fed children 565 who were dyspeptic or losing in weight; with this introduction the author refers to the successful **use of buttermilk** in Holland as a food for infants, and gives notes of 16 cases of malnutrition, selected from a large experience, with uniformly gratifying results, as long as the buttermilk was made from clean whole milk or cream; care in selecting the source of the buttermilk is urged, and the directions are to add to a liter a level tablespoonful of rice, barley, or other cereal, and boil, with frequent stirring, over a moderate fire for 25 minutes, then adding 2 or 3 heaping tablespoonfuls of cane- or beet-sugar (a porcelain or an enameled vessel free from lead should be used, as the acidity of the buttermilk is sufficient to dissolve lead if present); the buttermilk so prepared has a light yellow color and contains small curds, slow cooling producing smaller lumps than rapid cooling, these lumps necessitating a larger hole in the nipples; wretched, emaciated infants when given this nourishment cease crying and vomiting, take large amounts of it at a time, have 2 or 3 yellowish and soft movements a day, and gain steadily and rapidly in weight; the only possible disadvantage is a mild degree of rachitis, never more than an open fontanel and delayed dentition, and the author thinks that this can be attributed to other causes; the relation to metabolism is discussed at length. Salge[1] is also enthusiastic with regard to the value of buttermilk, although he states that in a small percentage of cases it may not agree, and other methods of feeding may be successful; he emphasizes the same points as de Mattos with reference to the source and cheapness; he finds that it contains 0.5 % to 1 % of fat, 2.5 % albumin, and 5 % sugar.

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[1] Ibid., vol. XXXII.

[2] Phila. Med. Jour., Sept. 14, 1901.

[3] Vratch, Sept. 1, 1901.

[4] Arch. of Ped., Nov., 1901.

[5] Arch. of Ped., Oct., 1901.

[6] Jahrb. f. Kinderheilk., vol. LV, 1902.

R. C. MacDonald[2] advises [as did Cotton, *vide* YEAR-BOOK for 1900] the use of **egg-albumen in milk-mixtures** to make up for the deficiency in lactalbumin, which is too low at the start and is lessened in amount by the dilution; the egg-albumen is also valuable as it is rich in organic phosphorus compounds.

Reinach[3] estimated the **fat in the feces** of 2 infants 6 and 9 months old, and then fed them with a preparation of chocolate, the fat of this being well absorbed, 1 child increasing rapidly in weight; having tried it in a number of cases, he gives as the indications inability to digest the fat of milk and a state of nutrition demanding an excess of carbohydrates and fat; it is also useful in acid dyspepsia, chronic enteritis, fat dyspepsia, failure to gain at the normal rate, and rachitis or scrofula.

H. L. Coit[4] reviews the epochs in the subject of infant-feeding, referring to Meigs' work in 1882, Rotch's in 1890, and Westcott's in

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[1] Ibid.

[2] Boston M. and S. Jour., Nov. 21, 1901

[3] Münch. med. Woch., No. 37, 1901.

[4] Arch. of Ped., May, 1902.

1897; Meigs, by his analysis of woman's milk, laid down the principles of substitute feeding, Rotch elaborated the percentage method as carried out in the laboratory and Westcott devised formulas by which the laboratory methods are rendered applicable outside of the laboratory. [The author modestly fails to mention his own important work in showing the profession their duty and the way to obtain certified milk.] The author refers to the failure of the profession to take hold of the subject of infant-feeding understandingly, and shows its importance, inasmuch as 50 % of children must be raised on the bottle; the difficulties of the subject are reviewed, the progressive principle in feeding is emphasized, and the author shows how the decimal system devised by him [*vide* YEAR-BOOK for 1899] renders this easy.

T. S. Southworth[1] discusses at length the **dietary for the second year** of life. J. E. Winters[2] takes up the subject of the food factor **as a cause** of health and disease during childhood, and mentions the principles by which the food should be adapted to the needs of the growing organism, calling attention, among other points, to the importance of giving a food necessitating chewing as soon as there are sufficient teeth, in order to develop the masticatory organs, including the salivary glands; he believes that **after the weaning period cereals and vegetables should be given abundantly** and flesh foods sparingly; he condemns the use of beef and beef-juice before a child is 2 years old, and recommends that between 2 and 5 years meat should not be given more than once in 2 days.

T. M. Rotch[3] includes as diseases of nutrition in infants infantile atrophy, infantile scorbutus, rachitis, and osteomalacia; the last one being the only one not characteristic of infancy; while the etiology is obscure, the author believes that all would be eradicated if the **diet of infants** was **properly regulated** and less medicine given. G. Alessandrello[4] theorizes as to the cause of **athrepsia,** holding that it is a chronic intoxication of gastrointestinal origin. J. L. Morse[5] discusses the **differential diagnosis of infantile atrophy from starvation,** wasting secondary to functional or organic disease of the stomach or intestine, congenital syphilis, and disseminated tuberculosis.

H. Cramer[6] studied the **metabolism** of 5 infants, 3 being newborn, by means of frequent weighings, which he believes give an exact method of determining the growth; he shows that the loss of water in the first few days of life causes an oliguria both absolute and relative to the amount of the ingesta, so that even when the ingesta remain the same, the daily amount of urine increases steadily during the first week; after the tenth day the daily amount of urine is about 70 % of the fluid drunk; the gas-forming excretory products in the newborn are subject to wide variations in short spaces of time, and the daily amounts are absolutely and relatively less than in older infants, there being an increase of elimination in the first 10 days of life.

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[1] Ibid.
[2] Med. Rec., Jan. 25, 1902.
[3] Med. News, July 13, 1901.
[4] La Pediatria, vol. x, No. 1.
[5] Med. News, Sept. 14, 1901.
[6] Arch. f. Kinderheilk., vol. xxxii.

Löffler[1] and M. Klimmer[2] discuss the hygiene of the dairy and the supervision of the production of milk. W. B. Ransom[3] sees no objection to boiling milk for 10 or 15 minutes provided it is used within 24 hours; in very hot weather the time of boiling should be half an hour, and the process should be repeated in a few hours if the milk is not consumed; in this way the spores of Bacillus sporogenes enteritidis may be destroyed. [The remaining articles on milk will be considered in connection with the subject of tuberculosis.]

INFECTIOUS DISEASES.

Tuberculosis.—S. A. Knopf[4] believes that the observations and experiments of Lartigau, Benda, Walter, Straus, von Leyden, Nocard, and others prove that heredity in tuberculosis is not a direct transmission of the bacillus, but a predisposition, and that all tuberculosis in childhood comes from without and not from within; the frequency of the disease in children, with its high death-rate, the maximum of which is marked between the second and fourth years, shows the need of children's sanatoriums for the prevention of the disease. Escherich[5] also urges the establishment by the State of **sanatoriums** where **convalescent** children from the hospitals could be sent for the development of a robust state of health before returning to their homes, which are too often unhygienic. C. Scott[6] writes of the importance of preventing tuberculosis in babes born of tuberculous parents, and cites 2 cases of maternal tuberculosis; one child was kept strictly away from the mother and is perfectly healthy at 3 years of age; the other was nursed by its mother for 4 weeks and died of tuberculosis at 17 weeks, 1 week after the mother. J. A. Robinson[7] discusses the **prevention** of tuberculosis in predisposed children, considering them in the three groups of infancy, childhood, and puberty; in infancy a good pure milk must be given; during childhood an abundance of nitrogenous food is desirable; at puberty the development of the mind and body should be harmonious.

Dieudonné[8] shows that children **creeping on the floor** may not only become infected with tubercle bacilli, but may be sources of danger to adults; he took 15 children living in houses with actively tuberculous adults and rubbed their hands and nasal cavities with cotton wads; these wads were then soaked in bouillon, and cultures, inoculations, and stains were made with 2 positive results.

C. H. Cattle[9] urges that a broad view be taken of the **transmission of tuberculosis to children,** bearing in mind the frequency with which they develop catarrhs of the respiratory tract, especially in connection with measles, etc., which render them susceptible to the widespread bacilli of human tuberculosis, these germs, whether introduced in air or food, affecting the most susceptible organs; English experience does not agree with Koch's statement.

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[1] Deut. med. Woch., No. 51, 1901.

[2] Jahrb. f. Kinderheilk., vol. LIV, 1901.

[3] Brit. Med. Jour., No. 2147.

[4] N. Y. Med. Jour., Nov. 30, 1901.

[5] Third Annual Report of the Hamidié Children's Hospital, Constantinople, 1902.

[6] Jour. Am. Med. Assoc., Sept. 28, 1901.

[7] Ibid., Aug. 24, 1901.

[8] Münch. med. Woch., No. 37, 1901.

[9] Brit. Med. Jour., Feb. 22, 1902.

Barthel and Stenstroem[1] studied **the influence of high temperatures on tubercle bacilli in milk,** different observers having had different results, which the authors attribute to the varying reaction of milk; an alkaline reaction, which is stronger as the disease of the udders is more advanced, renders sterilization very difficult. L. Rabinowitsch,[2] in treating of the infectiousness of the milk of tuberculous cows, emphasizes the fact that the only reliable method of examining the milk for bacilli is by intraperitoneal injection; this shows that the presence of tubercle bacilli does not, as a rule, correspond to the extent of the disease in the animal; clinical diagnosis of tuberculous disease of the udder does not mean that the milk always contains many bacilli, while, on the other hand, what may seem to be a simple mastitis may be associated with the presence of many bacilli in the milk; the author assigns great value to the tuberculin test in stamping out tuberculosis in cattle. E. S. St. B. Sladen[3] claims that heating, to 85° C., milk from tuberculous cattle will render it safe for use as a food; the clinical evidence in favor of the transmission of tuberculosis by milk is quite strong, and several instances are mentioned pointing to this; the author also believes that the frequency of tuberculosis after the infectious diseases like measles, etc., is largely dependent on infected milk.

Grawitz[4] discusses the **point of entrance** of the bacilli, and states that while the gross anatomic diagnosis points to a very small percentage of cases in which alimentary infection through the mesenteric glands is clear, it is not justifiable to infer that all other cases are of respiratory origin; he mentions that the intestinal mucosa is not very susceptible to the action of the bacillus as shown by the length of time that tuberculous sputum is swallowed before the consumptive by auto-infection develops intestinal lesions; the author has frequently found the tonsils, even when apparently healthy, to contain tubercle bacilli and giant-cells, the infection undoubtedly being alimentary; from the tonsils the bacilli would pass through the cervical glands and affect some of the thoracic organs apparently primarily.

M. Nicoll, Jr., and A. J. Lartigau[5] examined 75 **adenoids** removed from otherwise healthy children, taking one-half of each specimen for microscopic study and the other half for inoculation experiments; in 10 % both tubercle bacilli and the histologic lesions of tuberculosis were found, and in 5.3 % only tubercle bacilli were present; from the location of the bacilli the authors conclude that infection of adenoids takes place from the surface, apparently without any break in the epithelium.

A. Jacobi[6] points out that although primary intestinal and mesenteric tuberculosis is rare, yet the tubercle bacillus does not always produce lesions in the tissues through which it passes, and **peritoneal tuberculosis is common,** being in many cases of intestinal origin; the author firmly believes that milk from tuberculous cows should not be marketed.

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[1] Centralbl. f. Bakt., Oct. 8, 1901.

[2] Lancet, No. 4074.

[3] Lancet, Aug. 10, 1901.

[4] Deut. med. Woch., Oct. 10, 1901.

[5] Arch. of Ped., Aug., 1901.

[6] N. Y. Med. Jour., No. 1208.

H. L. Russell[1] considers the question from the standpoint of animal industry and public health; the first point requires that all herds shall be kept free from tuberculosis; on the second point, he believes that there is greater danger to human beings from tuberculous cattle than would be indicated by the lessened susceptibility of cattle to bacilli from human tuberculosis; with regard to the control of milk from cows reacting to tuberculin, he refers to Theobald Smith's work, which showed that pasteurization at 60° C. for 10 minutes entirely destroyed the bacilli. The question is also discussed by J. J. Repp,[2] J. McFadyean,[3] and E. W. Hope,[4] all of whom favor continuation of the present line of prevention.

P. Haushalter and A. Fruhinsholz[5] found in 78 autopsies in miliary tuberculosis, caseous enlargement of the tracheobronchial glands 74 times; in 67 the meninges were involved and in 63 of these the bronchial glands were affected; in half of the cases the adenopathy was the only old lesion, the lungs, if affected, showing only fresh miliary tubercles; the advanced caseation of the bronchial glands is explained by the view that the children were subject to repeated infections of small doses of bacilli; the authors refer to the established fact that in miliary tuberculosis the bacilli are disseminated by the blood-current, but in their researches the bronchial glands were always distinct, the walls of adjacent vessels being undisturbed; they raise the question of an infection from the glands intermediately through the lymphatic vessels, and so into the great veins; as regards the few cases of adenopathy and meningitis without tuberculous lesions elsewhere, Marfan has suggested an **infection through the lymphatics** by a retrograde current; this needs elucidation. J. Raczynski[6] contributes an interesting article on the **frequency and distribution** of tuberculosis in children, with remarks on the diagnosis; his statistics cover 611 cases in 3341 autopsies, or 18.3 %, the only other disease surpassing it in the period covered in the hospital being diphtheria; the mortality from tuberculosis was least in the first year of life, but increased steadily for each month of that year; half of the cases under 3 years of age were generalized tuberculosis, while over that age the lesions were more limited to the lungs and bronchial glands; the youngest patient was 21 days old, but in no case was there evidence of direct inheritance of the bacilli; in 2 cases there was apparently a primary intestinal infection, the mesenteric glands being tuberculous in a total of 67 cases; the author mentions the fact that some cases might be overlooked, as the bacillus occasionally lodges in tissues without setting up changes in them, their presence being detected only by careful microscopic examination or by inoculation experiments [of which we have seen one instance]; with regard to the diagnosis, out of the 611 cases 100 were unrecognized during life, illustrating the diagnostic difficulties, which the author then discusses, the first being that there is no regular type of tuberculosis in children and the bacilli cannot be easily demonstrated during life because the process rarely advances to destruction of lung-tissue and cavity-formation; emaciation is not always an accompaniment and fever may be either present or absent; examination of the blood and urine and even the x-rays may furnish no help; enlargement of the lymph-glands is a sign of great value, but caution must be used in interpreting it; almost the only sure sign is choroidal involvement; tuberculin is of value only in feverless cases; injection of artificial serum is positive only in a part of the cases; inoculation of lower animals with the blood of patients gave negative results in the author's hands. G. F. Still[1] found tuberculous lesions in 269 out of 769 autopsies in children, 223 of these dying of tuberculosis, or 28.9 %, the great majority resulting apparently from **infection through the respiratory tract**; causes which favor this infection and which may account for the increased susceptibility of young children to the disease are the infectious diseases with respiratory catarrh, like measles and pertussis; the author discusses the prevention of respiratory infection. Hajech[2] gives hospital statistics of 3640 autopsies in children under 5 years, 36 % showing tuberculosis; the majority were females and few were under 1 year of age. D. Boviard[3] discusses **primary intestinal tuberculosis in children,** its frequency and the evidence of its relation to bovine tuberculosis; in reviewing the literature he finds that only the English reports show any considerable number of cases of primary intestinal tuberculosis, 18 %, while the New York statistics show only a little over 1 %; this discrepancy seems inexplicable, and the author finds that the evidence connecting tuberculosis in children with the consumption of milk from tuberculous cows is scant. The author[4] reports a case of primary intestinal infection in a boy 3 years old. M. Nicoll, Jr.,[5] reports a similar case in a child aged 2¼ years.

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[1] Phila. Med. Jour., Nov. 16, 1901.

[2] Amer. Med., Nov. 2, 1901.

[3] Lancet, Aug. 3, 1901.

[4] Id., July 27, 1901.

[5] Arch. de Méd. des Enf., Mar., 1902.

[6] Jahrb. f. Kinderheilk., vol. LIV, 1901.

D. Durante[6] states that in tuberculous peritonitis with effusion the fluid is rich in lymphocytes, while other forms of peritonitis show a preponderance of polynuclear leukocytes. [This holds, with few exceptions, for other serous membranes, especially the meninges and the pleura.]

Cautley[7] analyzed 27 cases of tuberculous meningitis with reference to the etiology and morbid anatomy, and found that 22 were under 5 years of age; only 5 had a family history of tuberculosis; in 23 the mediastinal glands were caseous, and in 4 of these the mesenteric glands also showed the same change; of the remainder, the condition of the glands was not noted in 2, the other 2 having tuberculosis limited to the meninges, 1 of these being associated with caries of the cribriform plate of the ethmoid.

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[1] Practitioner, July, 1901.

[2] Jahrb. f. Kinderheilk., vol. LV, 1902.

[3] Arch. of Ped., Dec., 1901.

[4] Ibid.

[5] Ibid., May, 1902.

[6] La Pediatria, vol. IX, No. 11.

[7] Ref. in Arch. of Ped., Feb., 1902.

Rocaz[1] reports a case of meningitis probably tuberculous with recovery; the father of the patient, a boy of 8 years, had recently died of pulmonary tuberculosis, and the boy had many symptoms suggestive of tuberculous meningitis, with involvement of the lungs also; Arloing and Courmont's serum reaction for tuberculosis was positive and the cerebrospinal fluid drawn by lumbar puncture showed an excess of lymphocytes; after recovery a second tapping was done and the fluid was free from cells.

Dubief and Rabot[2] report a case of **tuberculous meningitis** in an infant 2 months and 20 days old; the tuberculosis was general, with the oldest lesions in the cervical and tracheobronchial glands. G. Gazal[3] reports a case in a girl infant, 22 months old, the symptoms being those of a left-sided hemiplegia.

H. Hellendall[4] recommends the intraspinal injection into guinea-pigs of cerebrospinal fluid from cases of suspected tuberculous meningitis, a general miliary tuberculosis being set up so quickly in the animals as to be of diagnostic value. J. K. Friedjung[5] has seen no therapeutic benefit from lumbar puncture in tuberculous meningitis, and as a diagnostic measure the bacilli are found in too small a percentage of cases and too late to do more than confirm the already clear diagnosis. [We have seen the procedure followed in a few cases by cessation of convulsions and vomiting, and in many others by evident increase in general comfort, while it has established the diagnosis in the majority of cases.]

Marcou-Mutzner[6] claims that an **excess of lymphocytes** does not prove a case to be tuberculous, as these were found in fluid from a man with fracture of the base of the skull; in fluid from a case of miliary tuberculosis of the meninges polynuclear leukocytes were in excess.

A. Walker[7] reports the administration of **large doses of creasote** (36 minims daily) to 2 children, 3 and 7 years old, afflicted with bone-tuberculosis; neither benefit nor harm seemed to follow.

B. K. Rachford[8] speaks of tuberculosis in infancy and childhood as being **essentially a disease of lymphatic structures,** and while it may be concealed or latent, yet there are often signs by which its existence may be suspected; the so-called scrofulous types, anemia without apparent cause, loss of or failure to gain in weight, tendency to take cold, and indigestion are some of the symptoms; the prognosis is better in children than in adults, the medical treatment which the author prefers being inunctions of guaiacol in lanolin and lard.

F. M. Crandall[9] points out that when **marasmus** depends on tuberculosis there are no characteristic signs by which the latter can be recognized.

Moizard and Bacaloglu[10] report 2 cases of **genital tuberculosis** in boys 15 months and 3½ years old; in the first case there was involvement of the right testicle and epididymis, the lesions being old, and of the seminal vesicle of the same side and of the prostate; death resulting from miliary tuberculosis involving the meninges; in the other case the testicles were healthy, but the kidneys, ureters, bladder, prostate, seminal vesicles, and ureters were extensively affected; the authors refer to the importance of rectal examination in the diagnosis. J. L. Duenas[1] reports a case of tuberculosis of the uterine appendages with extension to the peritoneum in a colored girl 11 years old; the symptoms were those of tuberculous peritonitis, and a laparotomy was done after several tappings, when the ovaries and tubes were seen to be highly tuberculous and were removed; the author considered the infection to have been through the blood, as the uterus and vagina seemed perfectly normal; there was reaccumulation of peritoneal effusion after the operation, but this yielded to treatment and recovery was complete.

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[1] Arch. de Méd. des Enf., Dec., 1901.
[2] Rev. mens. des Mal. de l'Enf., Dec., 1901.
[3] Arch de Méd. des Enf., April, 1902.
[4] Deut. med. Woch., No. 13, 1901.
[5] Wien. klin. Woch., Oct. 31, 1901.
[6] Arch. Gen. de Méd., Sept., 1901.
[7] Arch. of Ped., July, 1901.
[8] Arch. of Ped., Dec., 1901.
[9] Arch. of Ped., Jan., 1902.
[10] Arch. de Méd. des Enf., Oct., 1901.

S. S. Adams[2] reports 8 cases of pulmonary tuberculosis in the terminal stage in colored children, the daily **oscillations of temperature** being very great, 1 case showing a range of 10 degrees in 12 hours. [Some years ago we reported a similar case in a colored girl, the greatest range being 6.5 degrees.]

C. Beck and J. Grósz[3] report a case of **lichen scrofulosorum,** and discuss its relation to Darier's cutaneous tuberculides; in the authors' case the lesions showed round-celled infiltration with giant-cells, tubercle bacilli not being found.

Typhoid Fever.—J. H. Thursfield[4] believes that the **Widal reaction** is of greater value in children than in adults, and perhaps occurs earlier in children; a positive reaction is trustworthy evidence of the presence of typhoid fever, and a negative reaction after the tenth day points to the absence of the disease, while repeated negative tests are sure evidence [to which we have seen a few exceptions]; of 42 cases the reaction was positive in 40 on admission; and in the other 2 in the first week; none of the negative cases had typhoid fever. M. Gershel[5] found it positive in 81 out of 84 cases, while in 115 cases of fever not typhoid no positive reaction was obtained.

I. A. Abt[6] observed a case of typhoid fever in an **infant 21 months old,** in whom there developed an eruption on the back, neck, and extremities which was first papular, then pustular and gangrenous; death followed, and the autopsy showed an enlarged spleen, cholangitis, and adenoma of the liver.

R. J. Love[7] reports typhoid fever **in an infant 4½ months** old, the mother up to the tenth day of her own attack having nursed the child; he was then weaned, but was taken sick 2 weeks later with fever, rose-spots, diarrhea, and enlarged spleen, the disease ending fatally. A. Strasser[8] reports 2 cases of infantile typhoid fever. J. P. C. Griffith[9] states that **typhoid fever in children is often vague,** the course is shorter and the mortality lower than in adults; the onset may be sudden with vomiting and fever, or more gradual but without the malaise common in adults; the spleen is probably always enlarged, although not always discoverable; constipation is more frequent than diarrhea and nervous symptoms are more common in children than in adults. P. Galli[1] reports phlegmasia alba dolens complicating typhoid fever in a boy 5 years old. W. L. Rodman[2] operated on a girl 12 years old, 37 hours after **perforation in the fourth week** of typhoid, the patient making a good recovery. J. Sailer[3] reports typhoid fever **in a brother and sister,** both developing **noma** in the third week; diphtheria bacilli were cultivated in the necrotic areas and the antitoxin administered; the boy died, but the girl recovered; as a possible source of infection, the mother had had diphtheria some months earlier; another brother, who also had typhoid fever, did not develop noma, cultures from the throat not showing the diphtheria bacillus. H. E. Tuley[4] advises in the treatment of typhoid fever in children a liquid diet, including milk, koumyss, buttermilk, or broths, with plenty of water internally; hydrotherapy is the main reliance to combat the fever, flushing of the colon with cold water being also useful; further medication is sometimes unnecessary, but the author recommends bismuth subnitrate and tannalbin for the diarrhea, turpentine for distention, strychnin for cardiac weakness, alcohol being reserved until late in the course, when, if needed, it is to be given in large dose. L. d'Astros[5] observed a boy, 7 years old, who presented symptoms of perforation at the thirty-fifth day of typhoid fever; under absolute diet and opium, with injection of artificial serum, the symptoms subsided in 9 days, a relapse of typhoid occurring later with ultimate recovery, defervescence being complete on the seventy-first day.

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[1] Arch. of Ped., Oct., 1901.
[2] Arch. of Ped., Dec., 1901.
[3] Arch. f. Kinderheilk., vol. xxxiv.
[4] Brit. Med. Jour., Sept. 7, 1901.
[5] Med. Rec., Nov. 23, 1901.
[6] Jour. Am. Med. Assoc., vol. xxxvii, No. 7.
[7] Brit. Med. Jour., No. 2155.
[8] Med. Rec., Aug. 24, 1901.
[9] Jour. Am. Med. Assoc.. Aug. 17, 1901.

Diphtheria.—E. Müller[6] gives 3 charts which illustrate graphically the statistics of the cities of Germany having a population of 40,000 and over, with reference to the **mortality** from diphtheria between 1889 and 1900; there was a decided drop in the mortality in 1895, below the best of any preceding year, and the rate is being lowered steadily. F. Siegert[7] gives the statistics of the 5 children's hospitals in Vienna for diphtheria from 1886 to 1900, showing that the absolute as well as the relative mortality underwent a tremendous **reduction with the use of the antitoxin.** [These articles recall Virchow's remark of the "brute force" of the figures overcoming all objections.] J. A. Schabad[8] discusses the clinical bacteriology of diphtheria, believing in the distinction of the Klebs-Löffler diphtheria bacillus from the Hoffmann pseudodiphtheria bacillus, and stating that this is best made by the reaction of a bouillon culture and by Neisser's stain; he does not consider an avirulent diphtheria bacillus to be the pseudobacillus. This year it is A. Rupp[9] who disputes the specificity of the Klebs-Löffler bacillus as the real cause of diphtheria.

[1] La Pediatria, vol. IX, No. 11.

[2] Amer. Med., vol. II, No. 21.

[3] Am. Jour. Med. Sci., vol. CXXIII, No. 1.

[4] Jour. Am. Med. Assoc., Nov. 2, 1901.

[5] Soc. méd. des Hôp., July 19, 1901.

[6] Jahrb. f. Kinderheilk., vol. LV, 1902.

[7] Jahrb. f. Kinderheilk., vol. LV, 1902.

[8] Jahrb. f. Kinderheilk., vol. LIV, 1901.

[9] Med. Rec., Nov. 9, 1901.

R. L. Pitfield[1] describes a **double stain for the diphtheria bacillus** with the use of 3 solutions, A, B, C. The formulas are: (A) Silver nitrate 5 grams, distilled water 5 cc., saturated alcoholic solution of fuchsin 3 cc.; (B) pyrogallic acid 1 gram, 10 % sodium hydrate in water 5 cc., distilled water 10 cc.; (C) carbol-fuchsin solution 10 drops, distilled water 10 cc. After fixing the smear with heat, it is covered with solution A, which is boiled for a minute, and after another minute is poured off, and the slide treated in the same way, after washing, with solution B; after washing, solution C is applied for 1 or 2 minutes; the bacilli have a pink tint of varying depth, deeper where the protoplasm is heaped up, and at each end there are shining black points sometimes seen in the center; the cell-membrane has a slight grayish-brown tint. L. Cobbett[2] has devised a **modification of Neisser's stain,** consisting of 5 % acetic acid floated under the stained and mounted cover-glass; the bodies of the bacilli are then pale blue instead of brown; he believes that the stain is of very great help, the exceptions, when the true bacillus fails to show polar bodies, and when Hoffmann's bacillus shows them, being so few as to detract little from its value. R. M. Beaton, F. F. Caiger, and W. C. C. Pakes[3] investigated the **value of Neisser's stain** in 100 cases of angina, and feel that the stain is very valuable because the diagnosis may be made at once from a smear, because there seems to be no fallacy in the method, and because non-experts can recognize more surely diphtheria bacilli stained by this method than by methylene-blue.

J. Suesswein[4] found in 8 autopsies in diphtheria the **bacilli in the stomach** 4 times, but could not find them in the intestines; he attributes this to their destruction by the HCl of the gastric juice, whether free or combined; from studies on the vomitus during life he found the acid present in sufficient quantities; when the diphtheria bacilli get to the intestines, they are destroyed by the colon bacilli.

Cristeaunu and J. Bruckner[5] refer to the **rarity of diphtheria in the newborn,** and state that it is occasionally seen in maternity hospitals; they observed a small epidemic of 5 cases in infants a few days old, 3 dying in spite of antitoxin; the high mortality rests on the low resistance of the newborn child and on the difficulty of recognizing the disease until it has a start that cannot be overcome.

Caccia and Luigi[6] analyzed nearly 1000 cases of diphtheria, and found as a clinical feature that there is a certain amount of **antagonism** between **a localization of the disease** in the larynx and respiratory part of the pharynx on the one side, and the tonsils and pillars on the other side. [If we understand the authors rightly, exceptions to this are often so marked that dependence cannot be placed to any great extent on this protection.]

L. E. Holt[7] saw an infant 6 months old, with a **diphtheric conjunctivitis,** recovery being complete with the use of the antitoxin.

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[1] Univ. Med. Bull., Sept., 1901.

[2] Lancet, Nov. 23, 1901.

[3] Brit. Med. Jour., Sept. 21, 1901.

[4] Wien. klin. Woch., No. 6, 1902.

[5] Arch. de Méd. des Enf., Nov., 1901.

[6] Jahrb. f. Kinderheilk., vol. LV, 1902

[7] Arch. of Ped., May, 1902.

H. C. Beasley[1] reports a case of **relapsing diphtheria,** the membrane reappearing 10 days after the tracheotomy tube introduced in the first attack had been removed; antitoxin was used in each attack, but cultures were not made at any time.

Rabot and Bonnamour[2] call attention to the value of subcutaneous **injections of artificial serum in determining the prognosis** of infectious diseases in children, especially of diphtheria; they found that if, after the injections, the patient urinated freely without vomiting or diarrhea, the prognosis was favorable, no matter how ill the patient seemed; on the other hand, if the urine did not increase, but the injections were followed by diarrhea or vomiting, a fatal termination was to be expected, no matter how mild the case seemed; the authors state that the serum shows the condition of the kidneys and heart, and when these are profoundly affected, the water will not pass out through the kidneys.

After a discussion by Ausset, Barbier, Netter, Comby, and Sevestre,[3] the Paris Society of Pediatrics adopted a resolution affirming their belief that **immunizing injections of antitoxin** possess no serious danger, but confer immunity in the great majority of cases for some weeks, and recommending their use in institutions where children are grouped together and in families in which scientific observations cannot be had. H. D. Jump[4] believes that all persons exposed to diphtheria should be given from 250 to 500 units, according to age, and that this immunity will last for at least 3 weeks.

P. Geffrier and E. Rozet[5] recommend **small doses of diphtheria antitoxic** serum, rather than large doses, in order to avoid harmful effects of the serum, such as anuria, rashes, etc., of which they collect 13 reports; they use 5 or 10, occasionally 20 cc. [which, of course, conveys no idea of the dosage of antitoxin units; it would seem from this that a concentrated serum of 4 cc. containing 2000 units is not accessible in France]; in 309 hospital cases the authors have had the very good results of lowering a preantitoxin mortality of 28 %, to 6.47 %; the series included 137 laryngeal cases, of which 104 were operative; albuminuria was observed in 25 %, which shows, perhaps, a diminution as compared with the general idea of 50 % before antitoxin came into use; the chance of error in comparing statistics is possibly greater with reference to postdiphtheric paralysis, which occurred in 10 % of their cases, as contrasted with de Gassicourt's figures of 23 %; the disadvantages of serum-therapy lead the authors to prefer to rely on isolation, disinfection, and observation as measures of prevention rather than on preventive inoculations. G. Ritter,[6] of Rittershain, discusses the **serum-rashes,** and finds that they are now not only much less frequent (6.45 %, as compared with 22 % in 1897), but they are also much less harmful; the diagnosis of a scarlatiniform serum-rash is difficult, and quarantine had best be enforced unless a scarlatinal infection can be positively excluded; C. W. Bockley[1] reports a **rare form of purpura** complicating diphtheria; the patient was a girl 10 years old, and antitoxin was given on the fourth day, the membrane disappearing in 3 days; on the sixth day the patient vomited several times, with blood in the vomitus and in the stool; 2 days later deep purple papules appeared on the elbow, petechias came on the buttocks and later were seen on the face, ears, elbows, and knees; the papules on the elbows turned to vesicles which coalesced to form bullas containing a dark, bloody material; the joints were painful but not swollen; recovery was slow, and the author looks on the case as one of Henoch's purpura. [It certainly does not resemble the usual serum-rashes.] A. Stanley[2] found eruptions occurring in 112 out of 500 cases treated with the antitoxin; he describes the typical diphtheria antitoxin eruption as a marginate erythema in the psoriasis regions. [It is not accurate to speak of a diphtheria antitoxin rash, as the injection of serum which does not contain the antitoxin may cause the erythemas, and it has been clearly proved that the rashes depend not on the antitoxin but on the serum, and they should therefore be spoken of as serum-rashes.]

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[1] Lancet, No. 4070.

[2] Lyon méd., No. 34, 1901.

[3] Rev. mens. des Mal. de l'Enf., July, 1901.

[4] Phila. Med. Jour., Jan. 11, 1902.

[5] Arch. de Méd. des Enf., Feb., 1902.

[6] Jahrb. f. Kinderheilk., vol. LV, 1902.

J. J. Walsh[3] reports a case of **postdiphtheric urticaria** in a girl of 12 years; the attack of diphtheria was mild and antitoxin was not used; the author suggests that if the conditions produced by diphtheria were more carefully studied, the diphtheria serum might be freed from many suspicions attached to it. [We agree with this in general, but we do not believe that the author wishes to be understood as holding the opinion that the diphtheria serum does not cause the majority of cases of urticaria which are looked upon as serum-rashes.]

G. von Ritter,[4] of Prague, discusses at length **cicatricial contraction of the larynx following intubation.** . The various phases of intubation and tracheotomy in relation to diphtheria and the antitoxin treatment are brought out by J. von Bokay, J. Siegert, Pels-Leusden, G. von Ranke, Trumpp, and M. Pfaundler.[5]

A. N. Blodgett[6] recommends the **use of formaldehyd** during the course of diphtheria by hanging cloths wet with a 2 % solution in the room; the bacilli disappear from the throat-cultures quickly and the room becomes less infected. F. W. White[7] urges the **extension of quarantine** after diphtheria until 2 successive cultures from the throats of the patients and all associated with them have been negative; he observed a case of diphtheria develop as a result of infection by a healthy person who harbored the bacilli, the quarantine having been raised after one negative culture. [This is certainly a wise precaution, and one which ought to be followed in every instance, for we have seen several instances in which the bacilli were obtained again after one negative culture.]

Measles.—Diphtheria as a complication of measles is discussed by

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[1] Lancet, July 20, 1901.
[2] Brit. Med. Jour., Feb. 15, 1902.
[3] Phila. Med. Jour., vol. VIII, No. 15.
[4] Arch. f. Kinderheilk., vol. XXXII.
[5] Ref. in Arch. f. Kinderheilk., vol. XXXIII.
[6] Boston M. and S. Jour., No. 17, 1901.
[7] Ibid,. vol. CXLV, No. 9.

D. N. Blakely and F. G. Burrows,[1] who analyze 150 cases in the Boston City Hospital; the mortality was 34 %, while for uncomplicated diphtheria at the same time it was 13 %; the earlier in the measles the diphtheria developed, the worse was the prognosis; in a little over half of the cases there was membrane in the larynx, and 44 % of these died; of the 47 intubation cases, 55 % died; while of the laryngeal cases in which intubation was not done, 29 % died; the authors believe that diphtheria should be looked for in every case of measles, as the catarrhal condition of the upper air-passages predisposes strongly to a virulent action of the diphtheria bacillus if it gains a foothold. P. Armand-Delille[2] studied throat cultures from 75 cases of measles, finding in 12 % a bacillus morphologically and culturally like the Klebs-Löffler bacillus, but rarely possessing virulence; the virulent bacilli were almost always associated with symptoms of croup, but without animal inoculation the degree of virulence could not be determined, and the author therefore recommends in hospital and even in private practice immunizing injections of diphtheria antitoxin in all measles cases. J. Suesswein[3] reports an **epidemic of 21 cases** of measles, 10 of the cases having influenza bacilli in the respiratory discharges and 5 of them dying. C. Leiner[4] reports 4 cases of measles showing **blebs and bullas in convalescence** which the author decided to be **impetigo contagiosa** from the evident contagion, the feverless clinical course, the bacteriologic results, and the fact that autoinoculation produced typical impetigo. He considers that the cases of pemphigus complicating measles should be looked on as impetigo. P. Richter[5] treats of **pemphigus as a complication** of measles, and quotes extensively from the literature as far back as Rhazes (850) to show that its occurrence is not rare. It was apparently so frequent at one time as to need a differential diagnosis from variola. The author believes that it is a local condition brought about by a lowering of the resistance of the skin by measles, favoring the entrance of germs. A. A. Kissel[6] reports a case of **noma** following measles in a boy 5 years old, recovery ensuing although pneumonia and diarrhea with bloody passages existed. The latter numbered at one time as many as 60 a day. The noma progressed to loss of teeth and perforation of the lower lip, the bone being denuded. Treatment consisted in curetting with a sharp spoon, followed by the rubbing of iodoform in the parts and the use of potassium permanganate solution (1 : 4000) as a mouth-wash. A. Hall[7] reports a case of measles followed by **disseminated lupus** and death from tuberculous meningitis. J. E. Goldthwait[8] reports a case of **osteomyelitis** following measles. The tibia was affected, with destruction of the epiphyseal cartilage, so that after recovery the leg was shortened half an inch. Mauclaire[9] reports measles in a girl 12 years old, followed by osteomyelitis of the sternum

[1] Boston M. and S. Jour., July 25, 1901.
[2] Arch. de Méd. des Enf., April, 1902.
[3] Wien. klin. Woch., Nov. 14, 1901.
[4] Jahrb. f. Kinderheilk., vol. LV, 1902.
[5] Arch. f. Kinderheilk., vol. XXXII.
[6] Ibid.
[7] Brit. Med. Jour., Sept. 28, 1901.
[8] Ann. of Gyn. and Ped., vol. XIV, No. 15.
[9] Gaz. des Mal. Inf., vol. IV, No. 9.

and a mediastinal abscess which contained a quart of pus. After drainage the child recovered.

C. K. Bond[1] believes that while in some cases of recurrence of measles the primary attack was rötheln, nevertheless many instances are undoubtedly **true second attacks of genuine measles,** and he emphasizes the value of Koplik's spots in the diagnosis. [We agree with this fully.]

Scarlet Fever.—A. Baginsky and P. Sommerfeld[2] give an elaboration of their previous report [*vide* YEAR-BOOK for 1902] on the **presence of streptococci in fatal cases** of scarlet fever. Their present series comprises 82 cases, the heart-blood in every case giving cultures of a streptococcus which they describe fully. In using Class's culture-medium they did not observe the double form, like gonococci. Agglutination with the blood-serum of scarlatinal convalescents could not be obtained. The cultures varied in virulence, this variation not depending, in those obtained from the pharynx during life, on the severity of the disease. C. K. Millard[3] believes that the desquamation of scarlet fever is not infectious through all the period, but that the **scales may act as fomites.** Even before desquamation ends, some patients can mingle with safety with other people, and, on the other hand, some patients will be capable of spreading the disease even after peeling has ceased. J. B. Pile[4] suggests that the infection may be latent in the middle ear for a year, having seen a girl 14 years old, with **acute mastoiditis 1 year after** scarlet fever. There was a discharge from the ear, and 4 days later an older sister came down with scarlet fever, there being apparently no outside source of infection. F. Lommel[5] reports the development of scarlet fever in a child 12 days after having slept in a room in which, 233 days previously, a scarlet fever patient had been kept overnight. The room had been disinfected with formalin, and as the inmates of the institution (for deaf children) had no intercourse with the outside world, the author thinks it clearly proved that the contagion remained latent for that time. W. P. Williams[6] observed an epidemic of scarlet fever arising from **an infected milk-supply,** all of the cases being of a mild type and resembling rubella and Dukes's "**fourth disease.**" Some of the cases were placed in the scarlet fever ward with ordinary cases of scarlatina, but the former did not contract scarlet fever there nor did the latter get the "fourth disease."

F. P. Mackie[7] found that the blood in scarlet fever shows a **moderate anemia** constantly, and **also a leukocytosis** depending on the severity of the angina. An increase of the leukocytes seemed to be a good sign, and a sudden fall a bad one, as indicating beginning sepsis. The leukocyte count is of great value in the differential diagnosis from measles.

A. Baginsky[8] discusses thoroughly the varieties, symptomatology, prognosis, and treatment of **scarlatinal nephritis.** The paper shows evidence of an abundance of material, carefully observed and digested. Important clinical features of the complication are: (1) Temperature, the onset of nephritis usually occurring with a gradual, sometimes sudden, rise of temperature to 39° C. (2) The pulse often shows no relation to the temperature, but a wiry character is suggestive of uremia. (3) The amount of urine is lessened and the danger depends largely on the degree and duration of the lessening. (4) The morphologic elements should be examined from the 24 hours' amount, as single specimens may show great variations. (5) Dropsy, when extensive, is of bad omen, bronchitis and pneumonia being apt to develop. (6) The appearance of uremia is associated with a wiry, arrhythmic pulse, delirium, convulsions, aphasia, and hiccup. Of the 88 cases under observation, 11 were fatal, 5 having other complications; in 18 a chronic albuminuria persisted. In the discussion, Pfaundler referred to the frequent existence of albumosuria in such cases, pointing to a destruction of the body-albumin, and perhaps playing a part in the causation; Ritter thought that light cases of scarlet fever were more apt to be followed by severe nephritis than were the cases of severe onset, which, Baginsky replied, was due to the lack of care in the light cases; Zuppinger pointed out that nephritis would often develop in spite of rest in bed and milk diet.

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[1] Brit. Med. Jour., No. 2142.

[2] Arch. f. Kinderheilk., vol. xxxiii.

[3] Lancet, April 5, 1902.

[4] Lancet, June 15, 1901.

[5] Münch. med. Woch., July 16, 1901.

[6] Brit. Med. Jour., No. 2138, 1901.

[7] Lancet, No. 4069.

[8] Arch. f. Kinderheilk., vol. xxxiii.

Eichhorst[1] reports a case of scarlet fever in a girl 4 years old, followed by dry gangrene of the left leg necessitating amputation below the knee. The popliteal artery showed an endarteritis, and cultures from a left-sided otitis media and a furuncle on the forehead gave pure growths of Streptococcus pyogenes. Recovery ensued. The author reviews the literature of **gangrene following infectious diseases,** and finds among them but 3 cases due to scarlet fever (all of the left leg) and 1 due to diphtheria. R. Seubert[2] reports a case of gangrene of the left leg following scarlet fever, in a boy 7 years old; streptococci were found in the blood of the stump.

E. Fränkel[3] has seen 3 cases of **necrotic ulceration of the esophagus and stomach** following scarlet fever, 2 of the cases being extensive and 1 showing beginning invasion by the streptococci which cause the lesions and which enter from the mucous surface.

H. Illoway[4] observed apparently **permanent imbecility** follow an attack of scarlet fever in a boy 4 years old. The condition was considered the result of the general infection and not of a meningitis.

E. King[5] reports a case of scarlatina with the unusual nervous complications of **opisthotonus, trismus, and paroxysms of severe abdominal pain,** accompanying which were convulsions, cyanosis, and biting of the tongue. Recovery followed, and the author is unable to decide whether tetanus really existed or not.

Bouyer[6] reports a case of scarlatina in a girl 4½ years old in whom there occurred, 1 week after the onset, an attack of **hematuria, epistaxis, and petechias** lasting a week and ending in recovery. The author attributes it to a hemophilic tendency, as the child had nosebleed with measles a year earlier.

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[1] Deut. Arch. f. klin. Med., vol. LXX, 1901.

[2] Münch. med. Woch., No. 2, 1902.

[3] Virchow's Archiv, vol. CLXVII, No. 1.

[4] Arch. of Ped., May, 1902.

[5] Amer. Med., vol. II, No. 24.

[6] Arch. de Méd. des Enf., Jan., 1902.

J. M. Day[1] treats at length of the **diagnosis.** F. M. Crandall[2] refers to **joint-lesions,** the genuine strawberry tongue, and early nephritis as being among the more uncommon symptoms. L. Fischer[3] reports a case of **noma** in a girl 7 years old, following 3 months after scarlet fever. Recovery ensued with the use of hydrogen dioxid 50 %, the Paquelin cautery, and ichthyol. Vollmer[4] reports a case with the development of **pemphigus,** 4 weeks after the onset of scarlatina. Trommer[5] reports an epidemic of "**scarlatinosis,**" which resembles mild cases of scarlet fever, but in which the eruption appears first on the face and convalescence is established in 2 or 3 days without complications. [This recalls a statement to the effect that physicians formerly used the word "scarlatina" to describe a mild case of scarlet fever, as though it was a different disease from true scarlet fever.]

A. Seibert[6] lays stress on the **treatment of the nasopharynx in** scarlatina as being of the greatest importance so long as there is no specific remedy like the antitoxin in diphtheria. For superficial disinfection of the visible pharynx he recommends a carbolic-iodin solution (tr. iodin., 2.0; potas. iodid., 1.0; aq. dest., 120.0; acid. carbol., gtt. x), 1 teaspoonful of which may be given safely every hour for 4 or 5 days without toxic effects; it is to be swallowed while the child is lying on his back. For cleansing the nasopharynx the author again recommends irrigation with ichthyol solutions [YEAR-BOOK, 1901], and when these are insufficient he has used successfully in 6 cases a daily application of a 50 % alcoholic resorcin solution.

J. Grósz[7] reports on the **treatment of 168 cases** of scarlet fever, with a mortality of 19, or 11.8 %, excluding those moribund on admission. Of the fatal cases, 1 had bilateral empyema, 2 bronchopneumonia, 1 uremia, and 1 ulcerative endocarditis, while the remainder, 14, died of the septic form of scarlet fever. The treatment was symptomatic, high fever being occasionally combated with quinin or antipyrin if the heart was strong enough, but usually cold packs were used. Against the throat lesions, for which the author [sensibly] prefers the term scarlatinal necrotic angina to scarlatinal diphtheria, care was observed not to injure the mucosa, irrigations with 3 % boric acid, or 1 % potassium chlorate, being used in conjunction with cold or ice compresses. Sixteen cases were admitted with nephritis and 12 developed after admission, 1 of the former dying in a few hours, all of the others recovering. The treatment of this complication was that generally used—milk diet, digitalis, an alkaline diuretic, hot baths, and diuretin.

The report made by Clement Dukes [*vide* YEAR-BOOK, 1902], that he has seen in the boys at Rugby an **epidemic of what seemed to be neither scarlet fever nor German measles,** aroused much interest in all quarters, and met with varying comment. J. P. C. Griffith[1] believes that it tends to create confusion in an already cloudy area, while an editorial writer[2] holds that it may, if confirmed, tend to simplify matters, just as did the separation of measles from scarlet fever and of rubella from both of them. F. T. Simpson,[3] under the heading "Lamellar Desquamation in an Epidemic of German Measles or of 'Fourth Disease,'" reports a group of cases occurring in the American School for the Deaf in Hartford, Conn., the first 4 cases being diagnosed as rubella, which was revised later to scarlet fever on the discovery of lamellar desquamation; other cases developed, some of which were apparently typically scarlet fever and others rubella, and a return was finally made to the original diagnosis of rubella, for the following reasons: (1) Absence of albumin in the urine in all cases; (2) period of incubation, which was unquestionably fixed at 15 days or more [we must question this for some of the cases, as the dates given seem to show that 7 cases followed within from 3 to 9 days of the first]; (3) previous occurrence of scarlet fever in 5 cases, or 18 %; (4) profuse development of rash in all cases with slight disturbance of temperature or pulse, which would not occur in scarlet fever; (5) absence of vomiting, strawberry tongue, angina, and complications; (6) morbilliform type in two-thirds of the cases. Several of the cases which most strongly resembled scarlet fever had histories of a previous attack of that disease, and these cases, together with the others which showed lamellar desquamation, are considered by the author to have been instances of the "fourth disease" [but some of these cases have features which did not occur in Dukes's cases]; 4, and possibly 7, were reported to have had rubella, and the author states that "if we believe in the validity of Cullen's law that one attack of an eruptive fever entails immunity from a second attack during childhood [which undoubtedly has exceptions], we must regard it as demonstrated by the foregoing evidence that another variety of eruptive fever exists in addition to those already known." C. B. Ker[4] thinks that proof is not yet convincing. W. P. Williams,[5] in contradiction to Dukes, reports an epidemic of rubella in a school, there being 32 cases; some were of the scarlatiniform type corresponding to the fourth disease, but none of the other children contracted the fourth disease. J. H. Pleasant[6] reports an atypical epidemic of scarlet fever, and discusses Dukes's fourth disease, concluding that what Dukes saw must have been a mixture of scarlet fever and rubella. [As we have said in former issues of the YEAR-BOOK, this question will have a certain amount of obscurity until the etiology is definitely settled, and it would seem best until that time comes to be satisfied with the present clinical division of scarlet fever, measles, and rubella. Certainly the evidence for the existence of a fourth disease is far from convincing.]

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[1] Dublin Jour. Med. Sci., No. 363.
[2] Internat. Med. Mag., vol. XI, No. 1.
[3] Am. Jour. Med. Sci., vol. CXXIII, No. 4.
[4] Dermat. Zeitschr., vol. VIII, No. 2.
[5] Wien. med. Woch., No. 13, 1901.
[6] Arch. of Ped., Aug., 1901.
[7] Arch. f. Kinderheilk., vol. XXXIV.

J. Kramsztyk[7] reports 5 cases of **relapsing desquamating scarla-**

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[1] Phila. Med. Jour., April 12, 1902.

[2] Arch. of Ped., Sept., 1901.

[3] Ibid.

[4] Practitioner, vol. LXVIII, No. 11.

[5] Brit. Med. Jour., No. 2138, 1901.

[6] Phila. Med. Jour., May 24, 1902.

[7] Jahrb. f. Kinderheilk., vol. LV, 1902.

tiniform erythema, and mentions the difficulty of recognizing a single attack from scarlet fever. In 1 of his cases the attacks, 6 in 2 years, followed the eating of cheese. Points in the diagnosis are the relapses, the early desquamation before the rash has faded, and the large psoriasis-like plates that have several layers.

Pertussis.—Cima[1] has found that with the **leukocytosis** in pertussis there is a proportionate increase in the elimination of uric acid. L. Guinon[2] reports a case of pertussis in a girl 5 years old, followed by a widespread **multiple neuritis** of such extent that the child was utterly helpless and breathed only with the diaphragm; improvement set in and was uninterrupted, although scarlet fever and diphtheria followed. General **emphysema** complicating pertussis and catarrhal bronchitis was observed by H. C. Finch[3] in a girl 2½ years old, dyspnea and cyanosis being severe and death resulting in 6 days.

B. M. Basu[4] recommends the use of **tincture of cockroach** (Blatta orientalis) in doses of 3 drops every 2 hours; when the usual remedies, belladonna, bromids, antipyrin, etc., have failed, he has seen this remedy effect a cure in 3 or 4 days. T. J. Mays[5] attributes the paroxysms of pertussis to the action of a special **virus on the pneumogastric nerve,** and he therefore uses a line of treatment somewhat similar to that which he recommends for phthisis—counterirritation over the line of the vagus 2 or 3 times a day, with mustard, iodin, or a mixture of camphor, chloral, and menthol. In stubborn cases he injects 3 to 6 minims of a 2.5 % solution of silver nitrate, preceded by 5 minims of cocain solution of the same strength; he has seen marked amelioration in a day or two.

Influenza.—J. P. Soumaripas[6] states that **nephritis is not a rare complication** of influenza in children, and that frequent urine examinations are necessary during an attack, as the onset of the nephritis may be insidious. B. K. Rachford[7] reports 4 cases in patients between 4 and 12 years of age, 1 ending fatally; the onset was sudden, and the author does not consider the nephritis as a complication or a sequel, but as a part of the influenzal attack. D. J. M. Miller[8] reports a case of influenza in an infant 13 months old, **hemorrhagic nephritis** occurring as a complication, recovery ensuing; the author collects 40 cases of influenzal nephritis and makes a careful analysis. F. X. Walls[9] describes protracted influenzal pneumonia in infancy and reports 4 cases, lobar in character, the influenza bacillus being found in each case, in the glairy expectoration. Rendu[10] reports a case of influenza in a child 5 years old, developing on the fifth day **opisthotonus** and Kernig's sign; 5 days later the fever subsided, leaving a **flaccid paralysis** of the legs; lumbar puncture gave a fluid with increased albumin, cultures being negative. The author considered it to have been a case of cerebrospinal meningitis with coexisting acute anterior poliomyelitis, all of influenzal origin. H. Peucker[1] reports a case of influenza in a child 5 months old, with **pneumonia, pleurisy, and meningitis,** Pfeiffer's bacillus being obtained from all the lesions.

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[1] Jahrb. f. Kinderheilk., vol. LV, 1902.
[2] Rev. mens. des Mal. de l'Enf., July, 1901.
[3] Amer. Med., vol. III, No. 16.
[4] Indian Med. Rec., April 24, 1901.
[5] N. Y. Med. Jour., Sept. 7, 1901.
[6] Gaz. des Mal. Inf., vol. IV, No. 6.
[7] Med. News, vol. LXXX, No. 11.
[8] Arch. of Ped., Jan., 1902.
[9] Jour. Am. Med. Assoc., Aug. 31, 1901.
[10] Bull. et Mem. Soc. med. des Hôp., 1901, No. 4.

Vaccinia.—J. H. Huddleston[2] reports a case of generalized vaccinia in a male infant, 11 months old, a general eruption of discrete vesicles occurring 5 days after vaccination and turning to pustules in 3 days, without umbilication. Some physicians who saw it considered it to be varicella. Inoculation of the contents of some of the lesions into 3 other children of the family who had not been vaccinated nor had had varicella gave no result. The author collects 50 other cases from the literature, the majority having followed the use of humanized lymph.

Varicella.—J. F. Schamberg[3] describes an outbreak of varicella in children convalescent from variola, giving a discussion of the differential diagnosis. P. Nobécourt and P. Merklen[4] found no characteristics of the leukocytes in varicella which would be of diagnostic aid. Clear fluid from the blebs contained no cellular elements, while cloudy fluid had some red cells and mononuclear and polynuclear leukocytes. H. Dauchez[5] refers to the complications of varicella, especially nephritis, as being of importance, though rare. When present, they are the result of sepsis starting from the skin lesions, and the indication is therefore given to practice asepsis in every case.

Rheumatism.—S. Phillips[6] refers to 10 cases of epistaxis, all depending apparently on rheumatism, cardiac murmurs being present in half. The salicylates, which may cause nosebleed, had not been given, but their administration was begun with benefit. A. Josias[7] emphasizes the rarity of rheumatic nodes after first attacks of rheumatism, and reports a case in a boy 11 years old. The nodes were extensively distributed on the extremities and nearly all disappeared in 2 months. Histologic examination of 1 node showed a network of blood-vessels with thick walls, which the author compares to endocardial vegetations, both being probably due to the same infectious cause, which in this instance bacteriologic examination did not disclose. When nodes exist, endocarditis is always present, but the reverse does not hold. P. J. Barcus[8] reports a case of acute articular rheumatism in an infant 27 days old, the features being successive involvement of different joints, irregular fever, sour sweat, and recovery with the salicylates and alkalis. The author also believes that the heart was affected, owing to increased action, pulse of 125, and a slight displacement of the apex-beat. During the pregnancy the mother had rheumatism of the cervical muscles.

W. C. Bosanquet[9] reports a case of urticaria in a girl with a rheumatic history; some of the wheals were red and left brown stains due to a diapedesis of red corpuscles into the skin. Salicylates effected a cure.

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[1] Prag. med. Woch., 1901, No. 13.
[2] Med. News, No. 1495.
[3] Phila. Med. Jour., Mar. 8, 1902.
[4] Rev. mens. des Mal .de l'Enf., Aug., 1901.
[5] Arch. de Méd. des Enf., April, 1902.
[6] Lancet, No. 4095, 1902.
[7] La Méd. Moderne, Sept. 25, 1901.
[8] Arch. of Ped., Jan., 1902.
[9] Brit. Jour. of Dermat., No. 160.

Acute Adenitis.—C. Hochsinger[1] discusses glandular fever, and concludes that it is not a specific disease, but a regional adenitis, set up by infection in the distribution of the radicles of the glands. A. Guerin[2] reports 3 cases of glandular fever, which he considers merely a syndrome having a variety of causes. N. Fédele[3] reports an epidemic of 10 cases.

Tetanus.—J. S. Rawlins[4] reports a case of tetanus in a boy 10 years old, following a punctured wound of the foot. After an incubation of 6 days the disease developed rapidly, so that rectal feeding was necessary on the fourth day. Medication consisted in bromids, chloral for 6 days, then morphin and atropin. On the ninth day tetanus antitoxin was given every 6 hours for 4 doses with improvement in the condition, but convulsions on the twelfth day necessitated artificial respiration. Chloroform was given to the sixteenth day and morphin to the twenty-fourth, when convalescence was established.

Syphilis.—F. Scherer[5] reports 2 cases of Parrot's pseudoparalysis of congenital syphilis, which showed no involvement of the epiphyses, but streptococci were found in the central nervous system as well as in all the organs. The author believes that such cases may be due to the central action of the syphilitic toxin or of a bacterial toxin, as in his reported cases, the germs being evidently attenuated in virulence. C. G. Kerley[6] reports 6 cases of malnutrition due to congenital syphilis, and he believes that when the children of a family are puny, undersized, and with poor appetites and digestion, specific medication is indicated even if the family history is negative and the parents are in good health. [A generality which of course has many exceptions.]

Meningitis.—W. Hunter and A. W. Nuthall[7] found in 9 cases of posterior basic meningitis, occurring sporadically, a diplococcus identical with the meningococcus; in 3 cases the culture was pure, in 3 it was associated with the influenza bacillus, in 1 with the tubercle bacillus and in 1 with staphylococci. Busquet[8] proved by experiments on rabbits that the meningococcus when placed on the nasal mucosa will penetrate to the meninges, and cause meningitis. Zuppinger[9] points out that the **prognosis** of epidemic cerebrospinal meningitis in children **must always be guarded,** even when apparent convalescence has set in. He reports a case in a boy 7 years old with 4 relapses occurring at the third, tenth, seventeenth, and twentieth weeks, with marked improvement between them. Death occurred on the one hundred and fortieth day, the autopsy showing occlusion of Magendie's foramen with chronic internal hydrocephalus. Lumbar puncture was not done because its diagnostic aid was not needed, and at the time (1896) the author was not convinced of its safety. Sorgente[10] reports a case of meningitis in an infant 40 days old. A diplococcus, Jaeger's meningococcus, was obtained in cultures, and after studying it carefully the author believes that it varies so in its morphologic, biologic, and cultural peculiarities as to render the view justifiable that all diplococcal meningites are due to different forms of the same germ, those in which the germ closely resembles the pneumococcus being far more fatal than those in which it is like the meningococcus. E. Bendix[1] found an excess of lymphocytes present in 5 cases of tuberculous meningitis, but found the bacilli present only once in the fluid drawn by lumbar puncture. In 1 of 3 cases of epidemic meningitis due to the meningococcus there was also an excess of lymphocytes, which the author attributes to the chronicity of the course. L. Guinon[2] reports a case of pneumococcic meningitis in a hereditarily syphilitic infant 6 weeks old, pure cultures of the pneumococcus being obtained from the meninges and heart's blood. The entering portal could not be discovered. The case is of interest as showing that all meningites in syphilis are not necessarily syphilitic. X. Lewkowicz[3] discusses the **etiology** of meningitis and the **diagnostic value of lumbar puncture.** He divides meningitis into 4 clinicopathologic groups—epidemic cerebrospinal meningitis due to the meningococcus, simple meningitis due to varying infections, tuberculous meningitis, and noninfectious processes. Twenty-five cases are reported and tabulated on this plan. The meningococcus is described fully. In the tuberculous cases the lymphocytes were in excess in a majority of the cases, the author explaining the excess of neutrophiles in one case by the presence near the meninges of old, cheesy masses containing dead bacilli which have a positive chemotaxis.

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[1] Wien. med. Woch., Feb., 1902.

[2] Jour. de Méd. de Bord., June 30, 1901.

[3] La Pediatria, April, 1901.

[4] Memphis Med. Monthly, vol. XXII, No. 3.

[5] Jahrb. f. Kinderheilk., vol. LV, 1902.

[6] Med. News, Mar. 22, 1902.

[7] Lancet, No. 4057, 1901.

[8] Presse méd., Aug. 7, 1901.

[9] Arch. f. Kinderheilk., vol. XXXII.

[10] Jahrb. f. Kinderheilk., vol. LV, 1902.

Pneumonia and Pneumococcus Infections.—J. Comby[4] reports the case of a boy who had 2 attacks of pneumonia, the first when 3 years old, the second 6 years later; both times the right apex was affected, the signs being first demonstrable 3 and 5 days after the onset, the course each time being 9 days with high fever, although the area of consolidation was small. C. Leroux[5] reports the case of a girl 5 years old, dying suddenly on the third day with a pneumonia involving the right apex. S. Pearson and W. L. Carr[6] report a case of **general subcutaneous emphysema** complicating pneumonia in a boy 4½ years old; the emphysema remained at its height 5 days, during which the boy's condition was alarming, but improvement was gradual and recovery followed. The authors review the literature. Richardière and Delherm[7] report a similar case in a child 2½ years old, the bronchopneumonia following measles; dyspnea and cyanosis were absent and death occurred in 3 days, the autopsy showing rupture of dilated pulmonary vesicles.

G. Darling[8] gives the report of a patient, a girl 9 years old, having when first observed signs of **empyema and peritonitis.** The empyema was drained and a week later laparotomy was done, 1½ pints of greenish pus being evacuated. Recovery ensued. The author considers the case to have been one of pneumococcus sepsis. H. Ashby[9] reports a similar case in a girl 8½ years old, following pneumonia; 40 ounces of pus were drained from the peritoneal cavity and the empyema was evacuated at the same time. Recovery followed. The peritoneal pus contained Micrococcus tetragenus alone, while in the pleural pus it was associated with the pneumococcus; the latter had probably perished in the peritoneal pus in the 23 days before evacuation. G. Pfisterer[1] reports 8 cases of suppuration in connection with the bones and joints, caused by the pneumococcus, and tabulates 41 cases from the literature.

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[1] Deut. med. Woch., Oct. 24, 1901.
[2] Bull. et Mém. de la Soc. des Hôp., June 20, 1901.
[3] Jahrb. f. Kinderheilk., vol. LV, 1902.
[4] Arch. de Méd. des Enf., April, 1902.
[5] Ibid.
[6] Arch. of Ped., Feb., 1902.
[7] Presse méd., No. 104, 1901.
[8] Birmingham Med. Rev., No. 280.
[9] Lancet, No. 4103, 1902.

Erythema Nodosum.—A. Moussous[2] observed a case of erythema nodosum in the hospital, a similar case developing 10 days after the admission of the first, and he believes that among these erythemas there is a group which constitutes a specific infection, feebly contagious. F. Pauloch[3] reviews the subject at length, describing the symptoms and agreeing with Moussous.

Miscellaneous Infections.—M. Wassermann[4] points out that Bacillus pyocyaneus may at times become pathogenic, reporting 11 cases of **umbilical sepsis** in newborn infants due to the bacillus.

Zuppinger[5] reports 3 cases of death from heart failure in well-developed children, one having an abscess on the foot, one a superficial ulceration in the inguinal region, and the third having cervical abscesses. There were no symptoms of septicemia, but the autopsy in 2 showed **myocarditis and nephritis.**

R. G. Freeman[6] observed an epidemic in a foundling hospital affecting 77 children, all of whom were allowed to go to the playrooms in the basement. The symptoms were listlessness, anorexia, headache, and somnolence, and 12 children transferred to the hospital building had irregular fevers. The malarial organism was present in 1 case, but was not found in the others. Six grains of quinin daily cured all of the cases, and the author thinks that there is a possibility of the epidemic having been the **prodromal stage of malaria.** With this thought, an investigation showed that adjacent to the playroom was an excavation in which there had been stagnant water, but hardly a long enough time before the outbreak to allow of the development of the mosquito larvas, and, further, no mosquitos or bites were found.

G. N. Acker[7] reports a case of gonorrheal rheumatism in a colored girl 2 years old, with a **vulvovaginitis,** the discharge of which was rich in gonococci; recovery followed vaginal douches of boric acid, external applications of ichthyol, and the internal use of soda salicylate.

P. Nobecourt and P. Merklen[8] discuss the **part played by streptococci** in the **production of erythemas** frequently seen in infectious diseases, like typhoid fever, enteritis, etc. That streptococci have this faculty of producing erythema is shown by erysipelas. In 10 cases of various diseases in which erythema appeared the authors were able to obtain cultures of streptococci during life or after death in such numbers and locations as indicated them clearly to be the cause of the rash. Sometimes the evidence is not so clear, the infections being mixed. In the fatal cases the authors found such degenerative changes in the liver and kidneys that they conclude that, in the course of a streptococcal or other infection, when the liver does not destroy the toxins and the kidneys do not eliminate them, they can more easily influence the vasomotor centers of the skin and produce erythema or purpura.

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[1] Jahrb. f. Kinderheilk., vol. LV, 1902.

[2] Arch. de Méd. des Enf., July, 1901.

[3] Ibid., Oct., 1901.

[4] Virchow's Archiv, vol. CLXV, No. 2.

[5] Wien. klin. Woch., Aug. 22, 1901.

[6] Arch. of Ped., Feb., 1902.

[7] Arch. of Ped., Feb., 1902.

[8] Rev. mens. des Mal. de l'Enf., July, 1901.

Porak and Durante[1] report 4 cases of general **sepsis of umbilical origin** in newborn infants, and state that when the local symptoms are severe the infection is, as a rule, not dangerous. Severe sepsis may occur with almost no local change at the umbilicus.

L. D'Astros[2] discusses the causes of **osteomyelitis in infants under 3 months** of age, and reports 2 cases of a chronic type, spasm of the glottis occurring in each case, which the author holds depended on a chronic intoxication from the osteomyelitis.

Prevention.—C. V. Chapin[3] suggests that a more efficacious way of preventing contagion than the present conspicuously inadequate methods would be to educate each individual, starting with the teachers and scholars in the public schools, how to protect himself, especially with regard to books, pencils, slates, and the customs of spitting, wetting fingers, etc.

DISEASES OF THE ALIMENTARY TRACT.

Stomatitis.—E. E. Laslett[4] reports noma of bilateral development in a boy 6 years old, in the terminal stage of pulmonary tuberculosis. G. Blumer and A. MacFarlane[5] report 16 cases of noma occurring in an epidemic of measles; 9 cases were examined bacteriologically, cover-slips from all showing a thread-like organism in large numbers, which did not branch, were stained with carbol-fuchsin, and would not grow on any medium. Cultures gave the colon bacilli in all cases, usually with staphylococci and sometimes with streptococci; sections showed the necrotic tissue to be rich in the thread-like organism, the numbers diminishing as the healthy tissue was approached. The authors attribute these cases of noma to this organism, but do not hold that it is the specific germ in all cases. P. Israel[6] reports 2 cases of Fédé's disease (sublingual granuloma) occurring outside of Italy, only 2 others being on record. In 1 the lesions spread in spite of cauterization, and the child died. The growth was excised in the other and the patient recovered. The other 2 cases outside of Italy are reported by M. E. Audeoud,[7] cure following incision.

Tonsillitis.—J. Sobel and C. Herrman[8] report a series of 12 cases of tonsillitis due to the **fusiform bacillus of Vincent**; the patients were all children between the ages of 2 and 7 years, the attacks being associated with fever and a membranous deposit on the tonsils, soon becoming ulcerated; cover-glass preparations showed large numbers of fusiform bacilli and spirilla, the former being about twice as long as the diphtheria bacillus, pointed at the ends, sometimes crescentic and sometimes arranged end-to-end in a spiral or an L. The organisms did not grow on any medium. All the patients recovered, the duration of the lesions being about 3 weeks. E. Ausset and M. Dorion[1] state that acute primary inflammation of the pharyngeal tonsil is not rare in children, but is often overlooked, because adenoids, which are not necessary for the condition, are not present. The symptoms are high fever, and a dry cough, with no lesion to account for them, and the diagnosis can be made either by digital examination or by producing the pharyngeal reflex, when it will be found that the pharyngeal tonsil is enlarged and hard. Adenoids may result from repeated attacks.

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[1] Arch. de Méd. des Enf., June, 1901.
[2] Rev. mens. des Mal. de l'Enf., Nov., 1901.
[3] Med. News, No. 152.
[4] Lancet, No. 4095, 1902.
[5] Am. Jour. Med. Sci., vol. CXXII, No. 6.
[6] La Pediatria, vol. IX, No. 12.
[7] Rev. mens. des Mal. de l'Enf., vol. XX, No. 2.
[8] N. Y. Med. Jour., vol. LXXIV, No. 23.

Cyclic Vomiting.—A. B. Marfan[2] describes under the term **vomiting with acetonemia** attacks of vomiting affecting children between 1 and 10 years of age, occurring without fever and being uncontrollable. The attacks occur suddenly in apparent health, and may last 5 or 6 days. Acetone is often present in the urine and the odor of acetone is on the breath. Relapses may occur, but are not essential. The author thinks that it may be allied to cyclic vomiting. [It seems to us that the condition must be the same as that usually termed cyclic vomiting.] F. Valagussa[3] reports 4 cases and emphasizes the influence of heredity, the attacks being the childhood equivalents of gout.

Hypertrophic Pyloric Stenosis.—E. W. Saunders[4] reports 5 cases of pyloric stenosis in infants. In the first case vomiting began when the infant was 18 days old and death followed in 18 days, the autopsy showing hypertrophy of the circular muscular layer. This child was breast-fed, the mother's milk showing 4 % proteids. In the other cases vomiting began from 2 to 3 weeks after birth and responded to appropriate treatment, the author [properly] laying stress on the spastic nature of the disorder rather than on a congenital condition. The treatment recommended is: (1) medicinal, belladonna, bromids, and chloral being useful, opium being objectionable; (2) treatment of the secondary gastric irritation by lavage followed by rest of the stomach for 24 hours, nutrient enemas being given; (3) proper diet, which should leave no coagulum in the stomach, whey answering in this regard, to which peptonized milk can be cautiously added later. By placing the child on the right side after feeding, gravity will aid the motor power of the stomach. If improvement does not follow these measures, surgical intervention is to be considered. J. H. Larkin[5] reports a case in an infant, vomiting coming on at 2 weeks after birth and the child dying of marasmus 5 weeks later. At the autopsy the stomach was found dilated slightly and the pylorus was much thickened, there being microscopically an increase in the circular muscular fibers with replacement of the tubular glands of the mucous membrane by new connective tissue.

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[1] Arch. de Méd. des Enf., Aug., 1901.
[2] Arch. de Méd. des Enf., vol. iv, No. 11.
[3] Il Policlinico, 1901.
[4] Arch. of Ped., April, 1902.
[5] Ibid., Aug., 1901.

Gastroenteritis.—W. D. Booker[1] reviews the early writings on the subject of the summer diarrheas of infants, which began to appear after the middle of the eighteenth century, one of the earliest papers being by Benjamin Rush. C. G. Kerley[2] analyzes 555 cases of summer diarrhea among the out-patient poor, and emphasizes the fact that with proper care the mortality is not high, showing that the high infant death-rate during the summer is unnecessary. The author's treatment consists in withdrawing milk, giving a cereal decoction such as barley-water or later dextrinized gruel. The medicines used as indicated are calomel, castor oil, bismuth, and opium. The author decries the use of alcohol, albumin-water, and frequent irrigations. [The article is one of the best on the subject that have appeared for some time.]

Liver.—V. Immerwol[3] refers to Hutinel's statement that the most common form of cirrhosis is that accompanied by tuberculosis and symptoms of cardiac disease, the so-called cardiotuberculous cirrhosis, or, as Immerwol prefers, the pericardiotuberculous. The author reports 3 cases and observes that the stasis in the portal circulation is probably due to a compression of the inferior vena cava by bands of adhesion in the pericardium where the cava empties into the auricle. One of the cases had no cardiac symptoms, although marked lesions were found at the autopsy; another had all the symptoms of cardiotuberculous cirrhosis, but at the necropsy there was no symphysis of the pericardium and the enlarged liver with ascites was found to be the result of tuberculous glands in the fissure of the liver compressing the blood-vessels.

F. Passini[4] reports 3 cases of **cirrhosis of the liver** in children aged 6, 5½, and 4½ years. Microscopically there were seen acute and chronic changes, which seemed to indicate attempts on the part of the system to effect a cure. In the etiology alcohol could be excluded, but the author raises the interesting point that it is more difficult to exclude nonfatal accidental poisoning with phosphorus; he also refers to the possibility of intestinal infection being the cause. H. Audeoud[5] reports cases of hypertrophic cirrhosis of the liver in infants 14 and 15 months old; both were girls, and the course was rapid, lasting only 2 months, with jaundice and ascites. The author considers them to be instances of Hanot's disease and the result of infection from the intestines. G. Parker[6] reports 3 cases of congenital hepatic cirrhosis with obliteration of the bile-ducts, the patients living for 6 months, 7 months, and 5 weeks after birth. Jaundice was present at birth in 2 and appeared in the other in 3 weeks; ascites was marked in 1; and the liver was large in each, the autopsy showing imperviousness of the common duct and proliferation of the capillary bile-ducts. The author reviews the literature and refers to the obscure etiology of the affection. M. Wollstein[7] reports a similar case in a child dying at 3 months of age. C. Beck[8] reports a case of alcoholic atrophic cirrhosis in a girl 11 years old, and reviews the literature.

[1] Arch. of Ped., July, 1901.
[2] Arch. of Ped., Aug., 1901.
[3] Rev. mens. des Mal. de l'Enf., Aug., 1901.
[4] Arch. f. Kinderheilk., vol. xxxii.
[5] Arch. de Med. des Enf., June, 1901.
[6] Lancet, No. 4069.
[7] Arch. of Ped., Mar., 1902.
[8] Jahrb. f. Kinderheilk., vol. lv, 1902.

E. Schlesinger[1] reports a case of **primary cancer of the liver** in a boy of 4, with great enlargement of the liver and spleen, increasing cachexia and death 4 months after enlargement of the abdomen was first noticed. The author reviews the literature and tabulates the reported cases, finding 11 of secondary and 10 of primary (excluding his own) cancer of the liver in children.

Enteritis.—A Juergensohn[2] calls attention to some unusual causes of severe intestinal disturbances in infants, giving illustrative cases. A child of 6 months emaciated steadily and was thought to have mesenteric tuberculosis, but died with suppression of urine; examination of the new spring supplying the water which had been drunk showed it to contain nitrates and nitrites in abundance with traces of fat, probably from a neighboring graveyard which had been closed for 30 years. Two other cases are reported made ill by the water and 2 whose illnesses seemed to depend on the presence of antimony and free sulfur in the milk, evidently derived from the rubber stoppers of the Soxhlet apparatus, which are impregnated with a solution of these substances in the process of manufacture and are acted on by acid milk. Cleanliness of the apparatus and sweet milk effected a cure.

A. Baginsky[3] mentions the difficulty of securing proper material for a study of the **pathology of the intestinal tract** in infants, owing to the rapidity of postmortem changes. When these are avoided, it is then found that there are marked microscopic changes after the various forms of enteritis, even when the microscopic examination is negative. The author has found 2 varieties of changes—the catarrhal, affecting the mucous structures, and the follicular, affecting the lymphatic glands; but there are no clinical features by which these can be differentiated during life. After a thorough description of the morbid anatomy, the author discusses the etiology, and seeks to correct a misapprehension of his position by the French writers. He believes that all the summer diarrheas are saprogenous [putrefactive] in nature, whether of endogenous or exogenous origin, there being no specific germ at the bottom, but the ordinary saprophytic bacteria of the intestine having taken on a virulent action. In addition to the mouth as a portal of exogenous infection, he mentions the skin, and refers to streptococcal enteritis as following furuncles or suppurative middle-ear disease, the germs gaining entrance to the circulation and so affecting the intestinal wall. He believes that the testimony is against the reverse occurring, the complications of enteritis, such as bronchopneumonia, etc., being of exogenous origin, rather than from the blood infected from the intestine.

W. H. Robey[4] gives as the **treatment of infantile diarrhea** irrigations of the bowel twice daily, calomel in divided doses, and abstinence from food for 24 hours, using albumin-water with brandy. Further medication was unnecessary in the majority of the 172 cases forming the basis of the paper, and a return to milk-feeding, low percentages being given, was made in 24 hours. [From the characteristics of the types of cases we get the impression that summer diarrhea in Boston is a far milder disorder than in Philadelphia or New York.]

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[1] Ibid.
[2] Ibid.
[3] Arch. f. Kinderheilk., vol. XXXII.
[4] Phila. Med. Jour., July 27, 1901.

M. Pfaundler[1] reports results of an extensive **study of metabolism** in infants with gastroenteritis. The study was with special reference to Czerny and Keller's theory of acid-intoxication, the conclusion being that the increased elimination of ammonia by the kidneys is the result and not the cause of the special morbid conditions.

Appendicitis.—G. Blumer and H. Shaw[2] collect 8 cases of appendicitis in children under 2 years of age and report another in a 7 weeks' old child, athreptic and without special abdominal symptoms. At the autopsy, 48 hours after death, the colon bacillus was obtained in cultures, and smears showed cocci also in the pus around the perforated gangrenous appendix. The feeding had consisted of several proprietary infant foods. J. P. C. Griffith[3] gives the notes and autopsy findings in a case of appendicitis in an infant 3 months old, the symptoms being those of intestinal obstruction. He collects reports of 14 cases in children under 2 years of age. R. St.-Philippe[4] refers to the possibility of colic in infancy being located in the appendix. Sometimes the pain may come from the colon and be caused by stagnation of the contents, due very often to intestinal lithiasis.

Peritonitis.—J Comby[5] reviews the subject of peritonitis as a complication of vulvovaginitis in little girls, reporting 8 cases due to the gonococcus and pointing out that nongonococcal vaginitis rarely spreads to the peritoneum. He believes that the treatment of gonococcal peritonitis is purely medicinal, consisting in the application of ice locally and the use of morphin internally.

Intestinal Obstruction.—A. Wertheimer[6] treats of acute intestinal obstruction in childhood and reports several cases. One boy, 4 years old, was seized with abdominal pains, had convulsions, became apathetic, and died in 15 hours, the autopsy showing strangulation of the intestine by a Meckel's diverticulum the tip of which had become adherent to the mesentery. The second case was apparently an intussusception with spontaneous reduction. In the third case the diagnosis rested between reflex paralysis of the intestine and mechanical obstruction, relief following the free use of belladonna, opium having been given without success. Grange and Hau[7] report a case of stenosis of the sigmoid flexure and rectum following intussusception which had been spontaneously relieved by the passage of the intussusceptum, 12 cm. long. Intestinal obstruction developed some months afterward, and, the stenosis being unrelievable by operation, an artificial anus was established and the boy, 3½ years old, recovered. A. Primrose[8] reports a case of intussusception in a child 3½ years old, laparotomy on the third day being followed by recovery. F. W. Samuel[9] reports an autopsy in a case of intussusception, 6 invaginations being found in the lower two-thirds of the jejunum. E.

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[1] Jahrb. f. Kinderheilk., vol. LIV, 1901.

[2] Arch. of Ped., Aug., 1901.

[3] Arch. of Ped., Oct., 1901.

[4] Jour. de Méd. de Bord., Dec. 8, 1901.

[5] Arch. de Med. des Enf., Sept., 1901.

[6] Jahrb. f. Kinderheilk., vol. LV, 1902.

[7] Rev. mens. des Mal. de l'Enf., Dec., 1902.

[8] Canad. Prac. and Rev., vol. XXVII, No. 3.

[9] Arch. of Ped., May, 1902.

Weill and M. Pehn[1] report a case of congenital occlusion of the small intestine, the infant dying, although an artificial anus was made. For 15 cm. above the cecum the small intestine was a fibrous cord.

Dilation of the Colon.—E. Neter[2] discusses, with report of a case, the relation of congenital anomalies of the sigmoid flexure to dilation of the colon in childhood (Hirschsprung's disease) and to volvulus in adults. The author believes that the dilation of the colon is not congenital but depends on a congenital abnormal length of the sigmoid, an explanation of which may possibly be found in comparative anatomy. K. Tittel[3] reports a case in a boy 15 months old, the autopsy and histologic examination supporting the view of a congenital origin. M. Bjoerksten[4] reports a case in a girl 3½ years old.

Dysentery.—S. Amberg[5] found in the stools of 5 cases of dysentery in children Amœbæ coli containing red blood-corpuscles and associated with the presence of eosinophiles, of free eosinophile granules and Charcot-Leyden crystals. In 1 case eosinophilia was present in moderate degree, but no relation could be established between this and the number of eosinophiles and Charcot-Leyden crystals in the feces. When the crystals are found in the feces of children, there is a possibility of amebic dysentery. All of the cases recovered without liver abscess.

Prolapse of Rectum.—L. Baumel[6] states that the proper treatment of rectal prolapse is the medicinal treatment of the causal cough or diarrhea, and when this is relieved the prolapse will cease.

Rectal Polyps.—F. Huber[7] refers to a number of cases of rectal polyps, in children in whom adenoids also existed, and he believes that the polyps, like the adenoids, are a manifestation of the lymphatic constitution, the lymph-masses in the rectum being dragged on by their own weight and by the expulsive action of the intestine until a pedicle is formed.

Intussusception.—E. Owen[8] gives the pathology and diagnosis of intussusception, and states that the treatment is prompt abdominal section. In the discussion his position was upheld by D'Arcy Power, W. M. Eccles, A. H. Tubby, F. C. Abbott, J. P. Bush, S. White, L. Marshall, C. F. Cuthbert, A. F. Forster, E. M. Sympson, A. McPhedran, A. D. Blackader, F. E. Batten, and F. Eve, while B. Pitts thought it justifiable to use injections if the case were seen early.

DISEASES OF THE RESPIRATORY TRACT.

Diagnosis.—G. Variot and G. Chicotot[9] refer to the difficulties often experienced in attempting by the ordinary methods of physical diagnosis to map out the heart and to locate a pneumonia. They have found radiography of great value for these purposes. In outlining the heart allowance must be made for the divergence of the rays, and Chicotot has devised an instrument which makes the correction mechanically. Twelve cases of pneumonia were studied, in 6 the clear diagnosis being confirmed by the skiagraph, while the uncertain diagnosis in the remainder was made clear by the rays. In diffuse bronchopneumonia the picture is not so clear, and needs a practised eye to interpret it correctly. The authors recommend the use of a tube with rays of low intensity.

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[1] Presse méd., Aug. 17, 1901.

[2] Arch. f. Kinderheilk., vol. xxxii.

[3] Wien. klin. Woch., No. 39, 1901.

[4] Jahrb. f. Kinderheilk., vol. lv, 1902

[5] Bull. Johns Hopkins Hosp., No. 129.

[6] Arch. de Med. des Enf., April, 1902

[7] Arch. of Ped., Sept., 1901.

[8] Brit. Med. Jour., Sept. 7, 1901.

[9] Arch. f. Kinderheilk., vol. xxxii.

Adenoids.—Sarremone[1] refers to the importance of early removal of adenoids and gives a clinical picture of the condition, stating that the physician is apt to be disappointed by noticing no improvement immediately following the operation. Patience for a few months or a year is needed, when the child will present an entirely different picture.

Retropharyngeal Abscess.—S. V. Pearson[2] reports a case of retropharyngeal abscess in an infant 7 weeks old. The diagnosis is not difficult if the condition is thought of, laryngeal diphtheria being more often suspected at first. Hilton's method of opening from the side behind the sternomastoid muscle is recommended. [The minor operation of incising through the back wall of the pharynx, followed by prompt inversion of the child has given us such good results in the acute cases that it seems preferable to the major operation requiring anesthetization.]

Congenital Laryngeal Stridor.—Rocaz[3] reports 2 cases. One patient was a child 2 months old when first seen, laryngoscopic examination showing the epiglottis rolled on itself; this gradually unrolled, and the stridor had ceased by the fourteenth month except when the child cried. The other child was 8 months old, the epiglottis showing a folding on itself, and there was also hypertrophy of the arytenoepiglottidean folds. This child, whose mother was tuberculous, subsequently died of bronchopneumonia. The author gives the prognosis in such cases as being grave, owing to the predisposition to pulmonary complications. L. Cerf[4] reports a case in an infant, the stridor disappearing in the first month. The author mentions as causes other than the congenital malformation, adenoids, enlargement of the tracheobronchial lymph-glands or of the thymus, laryngitis due to aspiration of vaginal secretions during a slow labor (the prognosis of this form being grave), and a neurosis. As the stridor is only a symptom there may also be other causes.

Respiratory Failure.—I. M. Snow[5] reports 2 cases of respiratory failure in the newborn, one child being 4 and the other 7 days old. The symptoms indicated a bulbar origin, as the pharyngeal and anal reflexes were exaggerated, every attempt at giving food or medicine by the mouth or rectum causing cessation of respiration with cyanosis and general rigidity. Warm baths and inhalations of oxygen were used, one of the cases recovering. Reference is made to two similar cases in the literature. It is of interest in this connection to note the case reported by R. C. Buist[6] of an infant of 8 days with symptoms simulating opium-poisoning—pin-point pupils, cyanosis, and respiratory failure. Vomiting was absent, the pulse was good, but every attempt at swallowing made the breathing stop, necessitating artificial respiration. The temperature rose to 106.2° F. and the child died, the autopsy showing a pea-sized hemorrhage in the pons.

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[1] Rev. mens. des Mal. de l'Enf., July, 1901.

[2] Lancet, Oct. 26, 1901.

[3] Rev. mens. des Mal. de l'Enf., Feb., 1902.

[4] Arch. de Med. des Enf., Feb., 1902.

[5] Arch. of Ped., Oct., 1901.

[6] Yale Med. Jour., vol. VIII, No 3.

Persistent Laryngeal Stenosis.—J. P. C. Griffith[1] reports a case of very persistent laryngeal stenosis in an infant 20 months old. For 6 weeks before admission there had been marked dyspnea, but there was no pseudomembrane in the throat and cultures for the diphtheria bacilli were negative. Laryngoscopic examination showed redness and swelling of the vocal cords, and in 3 days the dyspnea became so great that intubation was done with immediate relief; the tube was worn for 18 days, being gradually removed without a return of the dyspnea either then or shortly afterward when pneumonia developed. The author discusses the differential diagnosis, concluding that the condition was a subacute laryngitis associated with a personal and family tendency to spasm, allied to laryngismus stridulus but differing in its persistence.

Gangrene of the Lung.—F. Huber[2] describes pulmonary gangrene in children and mentions 3 fatal cases and 2 cases that recovered. Of the latter, 1 followed traumatism, with possibly inspiration of a foreign body,—a peanut which was said to have been coughed up,—improvement following immediately. The other case was of unknown origin, and operation was performed, pus being discharged from the mouth during the operation. A portion of rib was removed, drainage inserted, and the boy recovered after several months. W. L. Carr[3] reports a case in an infant a year old, following pneumonia. The literature is reviewed.

Bronchitis.—R. St.-Philippe[4] describes a form of chronic bronchitis in children with râles like asthma but without the dyspnea, to which he applies the term emphysematous bronchitis and for which he recommends arsenic iodid in saturated solution, starting with 5 drops after each meal and increasing to 15 or 20 drops 3 times a day, continuing at the maximum dose for a month and then withdrawing it gradually.

Pleurisy and Empyema.—J. Carmichael[5] gives a clinical lecture on 3 cases of pleurisy. The first patient was a boy, 6 years old, who had formerly been treated for tuberculous peritonitis, 11 brothers and sisters having died of tuberculosis; the second time that he came under observation there was a right-sided serous pleural effusion, recovery from this being complete, the ultimate prognosis being doubtful but hopeful. The second case was a pneumococcic empyema in an infant 1 year old, recovery following drainage. In the third case a large serous effusion was absorbed rapidly under medicinal treatment. W. T. English[6] discusses the differential diagnosis of infantile pleurisy with effusion from pneumonia, urging aspiration whenever fluid is suspected. A. A. Kissel[7] refers to some good results in unpromising cases of empyema, obtained with a modification of Lewaschoff's method. A boy 2½ years old, much emaciated, with a seropurulent pleurisy of a month's duration and a tuberculous family history, recovered completely after aspiration at different times, the removed fluid being replaced by normal salt solution until this returned clear. A. Jacobi[1] places on record a unique case of empyema in a girl 7 years old, evacuation of the pus and resection of a rib being followed by profuse hemorrhage from tufts of granulations which were visible on the visceral pleura. The bleeding was controlled by packing with gauze, the case progressing to uninterrupted recovery. L. E. Holt[2] reports a case of high temperature of 3 months' duration in a boy 2½ years old, the physical signs being inconclusive except for a consolidation of the right upper lobe followed by râles equally distributed on both sides. At the autopsy there was increase of connective tissue in the right upper and left lower lobes, with general obliteration of both pleural sacs, and 2 sacculated empyemas, one on the right on the diaphragmatic surface and one on the left close to the spine near the base.

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[1] Arch. of Ped., Mar., 1902. [2] Arch. of Ped., Mar., 1902. [3] Ibid.
[4] Rev. mens. des Mal. de l'Enf., July, 1901. [5] Arch. of Ped., Sept., 1901.
[6] Med. News, Nov. 16, 1901. [7] Arch. f. Kinderheilk., vol. xxxii.

Sorgente[3] reports a case, the third in pediatric literature, of **chylous pleurisy** in a girl 7 years old. Repeated aspirations were necessary, 1890 cc. of fluid being removed in 5 months, recovery being complete. The author believes that there had been a tuberculous ulceration of a large lymphatic vessel.

Zuppinger[4] discusses the **etiology of pneumothorax** in children and reports a personal observation in a child 2½ years old taken suddenly ill during sleep with abdominal pain, dyspnea, and restlessness, death occurring in 36 hours. The cause was a sharp particle of wheat penetrating the pleura and which had probably been aspirated from a straw sack on which the child slept.

DISEASES OF THE HEART AND BLOOD-VESSELS.

D. B. Lees[5] discusses various points with regard to the heart in childhood, including the method of examination, the disturbances of the myocardium, and the treatment of the rheumatic heart.

P. Merklen[6] reports a case of **essential paroxysmal tachycardia** in a girl of 13 years, who has had 3 attacks, the last one lasting a week until leeches were applied to the precordia, when the pulse-rate fell suddenly from 220 to 80 beats a minute.

L. Morquio[7] reports a familial disease affecting nearly all the children in one family, with symptoms similar to **Stokes-Adams' syndrome,** characterized by slowness of the pulse, syncopal and epileptiform attacks, and sudden death. There had been 8 children in the family, the oldest and youngest still living at the ages of 17 and 2 years respectively, the fifth dying at 5 years of typhoid fever, none of these 3 having shown the attacks. In the others the attacks began at the fourth year, the second child dying suddenly at 10 years, the third and fourth at 8 years; the seventh child is subject to the attacks, being still alive at 5 years; the sixth died suddenly when under observation, and the autopsy revealed nothing abnormal except an enlarged thymus. The author attributes the condition to a congenital malformation or hereditary weakness of the cardiobulbar centers, as the usual lesions of Stokes-Adams' syndrome, fatty degeneration, or arterial sclerosis are absent.

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[1] Arch. of Ped., July, 1901.

[2] Arch. of Ped., Jan., 1902.

[3] Jahrb. f. Kinderheilk., vol. LV.

[4] Wien. klin. Woch., Jan. 2, 1902.

[5] Lancet, No. 4092, 1902.

[6] Jour. des Praticiens, May 25, 1901.

[7] Arch. de Méd. des Enf., Aug., 1901.

H. Roeder[1] contributes an interesting article on the **histogenesis of the ductus arteriosus,** and points out that its entrance at an acute angle, which is nearly constant at 33 degrees, is not the result of chance but is an expression of static and mechanical laws, which also govern the richness of its walls in muscular and elastic tissue, so as to obtain the greatest functional activity with a minimum of material. J. Esser[2] reports 2 cases of rupture of the ductus arteriosus in the newborn. The first infant was born with the cord wrapped around the neck, causing asphyxia, resuscitation being impossible; the second developed sclerema and died on the sixth day, the respirations having been very shallow. H. Roeder[3] found at the autopsy on an infant 10 days old that had been delivered by version and had died of sepsis, a solid, thrombosed, dilation aneurysm of the ductus arteriosus half as large as a cherry. Other effects of the tedious delivery were seen in overriding of the cranial bones and fracture of the clavicle.

G. Berti[4] defends his theory of the **blood-nodes on the cardiac valves of the newborn** against that of Haushalter and Thiry, who consider them to be the result of the changed direction of the blood-current and the increased pressure following the infant's struggles and cries, all of which force the blood into small emissaries or canals along the attachments of the chordæ in the marginal part of the under side of the auriculoventricular valves. Berti holds that they are covered with endothelium and are the result of ectasis and cyst-formation in the valve-structure itself.

J. L. Morse[5] analyzes 32 cases of **congenital heart disease.** The paper is one which cannot be abstracted, but the author brings out as interesting points that in a number of cases the condition was only discovered in routine examination, there having been no cardiac symptoms even up to the age of 4 years in several cases; that the symptoms from patent foramen ovale are comparatively mild, and that some cases recovered from lesions which were apparently the same as those which in others caused chronic invalidism or death.

T. Fisher[6] reports 2 cases of congenital disease of the left side of the heart, one lesion being mitral stenosis, the child dying at 15 months; the other was aortic stenosis, the child dying in the fifth month of bronchopneumonia. E. E. Graham[7] reports a case of congenital heart disease in an infant dying at the twelfth month, having been apparently well for the first 6 months of life, and then having attacks of cyanosis and dyspnea. The autopsy showed a deficiency of the membranous portion of the interventricular septum, a small opening in the foramen ovale, pulmonary stenosis, and the aorta arising from both ventricles.

[1] Arch. f. Kinderheilk., vol. xxxIII.

[2] Arch. f. Kinderheilk., vol. xxxIII

[3] Virchow's Archiv, vol. CLXVI, No. 3.

[4] Arch. f. Kinderheilk., vol. xxxI.

[5] Arch. of Ped., Oct. 1901.

[6] Brit. Med. Jour., No. 2150, 1902

[7] Arch. of Ped., Mar., 1902.

A. Zuber and P. Armand-Delille[1] report a case of **infectious endocarditis** in a boy 13 years old, the subject of 4 previous attacks of rheumatism. The aortic valves were most affected, and a coccobacillus was obtained similar to the one found by Litten in a nonseptic case of malignant endocarditis. G. Gabriel[2] reports a case of acute streptococcal endocarditis in a boy of 10 years, with cerebral embolism and with the development of an aneurysm of the superior mesenteric artery.

I. A. Abt[3] analyzes 22 dispensary cases and 50 hospital cases of **heart disease in infancy and childhood.** Of the ambulant cases, only 2 gave a distinct history of acute rheumatism, 20 had had rheumatic pains, 6 tonsillitis, 2 chorea, and 9 scarlatina. Mitral regurgitation was the sole lesion in 15 of the 22. Of the hospital cases, only 11 had pure mitral regurgitation, while 18 had a double mitral lesion. Pericarditis existed in 11.

A. D. Blackader[4] discusses **acute dilation of the heart** met with during childhood and adolescence. It may be seen during any of the infectious fevers or, in health, as a result of violent exercise. The treatment consists in rest in the recumbent posture, digitalis, strychnia, and alcohol or ammonia, as needed.

G. F. Still[5] discusses **suppurative pericarditis** in children, and states that the cases are usually in children under 3 years of age, the pneumococcus being the cause in the majority. In 769 autopsies 11 cases were found with pus in the pericardium, 17 others without pus giving cultures of pyogenic germs. The pericarditis is rarely the sole process, so that chance of recovery is not great, even with evacuation, which is best done by incision and resection of costal cartilage rather than by aspiration. F. E. Batten[6] points out that the diagnosis is very difficult, suggestive symptoms being a rapid pulse and syncopal attacks.

A. Baines[7] reports a case of **arteriosclerosis** in a boy 10 years old, all of the palpable arteries being hard and cord-like. The urine was of low specific gravity and contained albumin, and there were frequent attacks of bleeding from the gums and nasal mucosa. The autopsy showed an advanced interstitial nephritis, with general arterial fibrosis most marked in the smaller vessels. The boy had had measles and chorea, but otherwise his history was negative.

T. Fisher[8] reports 4 cases of **primary thrombosis** of cerebral veins and sinuses in children.

DISEASES OF THE BLOOD.

E. Schiff[9] investigated the **specific gravity** of the **blood of newborn children,** and found that while there were individual differences, there was a steady lowering during the first 10 days of life from 1.076 to 1.0652. The specific gravity in the daytime is higher than at night, the maximum difference being 0.003. Conditions influencing it are the degree of development of the infant and the method of treating the cord, the specific gravity being higher in well-nourished infants and when ligation of the cord is delayed. The sex of the child and whether the mother is a primipara or a multipara have no influences. Jaundice lessens the specific gravity. There seems to be no relation between the specific gravity and the number of corpuscles or amount of hemoglobin, but all seem to be affected by nationality.

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[1] Arch. de Med. des Enf., June, 1901.
[2] Wien. klin. Woch., No 43, 1901.
[3] Medicine, vol. VII, No. 7.
[4] Montreal Med. Jour., vol. xxx, No. 7.
[5] Brit. Med. Jour., No. 2123.
[6] Ibid.
[7] Arch. of Ped., Aug., 1901
[8] Brit. Med. Jour., July 6, 1901.
[9] Jahrb. f. Kinderheilk., vol. LIV, 1901.

Geissler and Japha[1] call attention to certain facts in connection with **anemia in children.** The principal change in the red cells is the appearance of nucleated forms. The normal number of leukocytes is usually above that for adults, and the proportion of lymphocytes is increased. The authors believe that a classification of the anemias of childhood cannot be rationally based according to the number of leukocytes, and that in each case the numeric value of each variety of the leukocytes should be given. Enlargement of the spleen cannot be made a differential point for the anemias, for it is found in the light as well as in the severe forms, and may be present without anemia. The term splenic anemia is suitable for those grave secondary anemias occurring in rachitis with enlargement of the spleen, the prognosis being not always unfavorable. The term pseudoleukemic anemia is evidently used by different writers to include very different conditions, ranging from the simple splenic anemia to leukemia. Reports of recovery from leukemia need the confirmation of further observations.

G. D. Head[2] discusses the **leukocyte count** in the diagnosis of diseases in children, and states his belief that in the first 2 years of life it is of value mainly in recognizing those diseases, like measles, tuberculosis, typhoid fever, influenza, and malaria, which are associated with a lessened number of leukocytes. After 2 years of age the leukocyte count gives as fixed results as in adults. The author tabulates counts of his own and other observers for a number of different diseases.

Pernicious Anemia.—T. M. Rotch and M. Ladd[3] make a preliminary report on a case in a female infant, 9 months old, who had been breast-fed for 4 months, and then, owing to loss of weight and intestinal indigestion, had been fed on modified cow's milk. At 5 months there was a mild attack of measles, after which her development seemed normal, with only occasional mild attacks of indigestion. A slight pallor was noticed at the eighth month, which became marked 2 months later, when the sunburn of summer had disappeared. Careful examination then showed a moderate range of fever with slight puffiness of the hands and feet, a soft systolic murmur all over the precordia and in the neck without cardiac enlargement, the urine having a very faint trace of albumin, but no casts. Blood-examination showed Hb, 30 %; red cells, 1,732,000; leukocytes, 9200; nucleated red cells, poikilocytes, and many macrocytes were present. The hemoglobin sank to 20 % and the red cells to 1,088,000, and a state of collapse developed. At this time inhalations of oxygen were begun, for 10 minutes, every 20 minutes day and night, and a coincident improvement was at once noticed, the Hb increasing to 35 % in 2 weeks, to 60 % in 4 weeks, to 70 % in 6 weeks, and to 80 % in 17 weeks from the start of the oxygen. All possible causes of secondary anemia were investigated and excluded. The authors precede their report by a careful consideration of the etiology and pathology as set forth in the literature, finding 6 other cases, all fatal, reported in infants under 2 years of age. [Defining pernicious anemia as an anemia with certain symptoms and a definite blood-picture and without discoverable cause, we see no reason why this case should not be accepted as such.]

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[1] Jahrb. f. Kinderheilk., vol. LIII, 1901.
[2] Arch. of Ped., April, 1902.
[3] Arch. of Ped., Sept., 1901.

Leukemia.—J. A. Scott[1] reports a case of chronic lymphatic leukemia in a boy of Italian parentage, under observation from the age of 9 to 21 months, when death occurred. There was great enlargement of the spleen with a leukocytosis, the lymphocytes steadily increasing. Attacks of diarrhea were frequent, and the author believes that splenic leukemia and v. Jaksch's pseudoleukemia infantum, of which the case is a type, are secondary anemias.

Rocaz[2] reports a case of acute lymphatic leukemia with **hypertrophy of the thymus** in a boy 4 years old. The onset was with an angina, following which the lymph-nodes enlarged, the red cells were reduced to 1,800,000, and the leukocytes increased to 245,000. Differential count showed 96.6 % lymphocytes, 0.6% large mononuclears, 6 % transitionals, and 2.4 % polynuclears, with absence of eosinophiles, myelocytes, mast-cells, and nucleated red cells. Death ensued on the twenty-fifth day, the thymus at the autopsy weighing 200 grams. S. Welt-Kakels[3] also reports a case in an infant 18 months old, the leukocytes numbering 682,000 to the cubic millimeter.

Secondary Anemias.—A. H. Wentworth[4] reviews the literature on v. Jaksch's **anemia infantum pseudoleukemica,** which has been described as a primary disease of the blood. The characteristic symptoms of enlarged spleen and severe anemia, often with leukocytosis, are considered by the author [rightly] to be secondary to a variety of obscure causes. T. Guida[5] also believes that the disease is a secondary anemia dependent on gastrointestinal disturbances, improper food, and especially disturbed assimilation. C. Herrman[6] reports a case in a girl 1½ years old presenting the typical picture as described by v. Jaksch. Morquio[7] reports a case showing improvement under treatment. L. E. La Fetra[8] reports a case of secondary anemia in a child 2 years old, sent with a **diagnosis of pernicious anemia.** A count showed Hb 30 %, red cells 210,000 [evidently, from the count 2 weeks later, a typographic error for 3,210,000], leukocytes 13,200, no poikilocytes nor nucleated red cells. Regulation of diet and small doses of iron were followed by a rapid recovery.

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[1] Am. Jour. Med. Sci., vol. cxxiii, No. 1.

[2] Rev. mens. des Mal. de l'Enf., vol. xx, No. 3.

[3] Arch. of Ped., June, 1902.

[4] Boston M. and S Jour., Oct. 31, 1901.

[5] Rif. med., vol. xvii.

[6] Arch. of Ped., Nov., 1901.

[7] Rev. Med. del Uruguay, May, 1901.

[8] Arch. of Ped., Nov., 1901.

D'Orlandi[1] reports a case of **splenic anemia** in a child 11 months old, showing improvement after 4 months' treatment with spleen-extract, as suggested by Cervesato in 1896, since which time 4 cases have been treated with it, all successfully. The reasons for this treatment are: (1) the hypothesis that the spleen has an internal antitoxic secretory function which is disturbed in this disease; (2) the failure of other forms of treatment; (3) the bad prognosis.

Hemophilia.—N. von Etlinger[2] reports a case of **fatal bleeding from the right conjunctival sac** in a 3 weeks' old infant; there were also bronchitis, vomiting of blood, petechias, and conjunctivitis.

CONSTITUTIONAL DISEASES.

Achondroplasia.—Escherich[3] demonstrated specimens from a case dying in the first year of life. He points out that the disease has nothing at all to do with rickets.

H. Ashby[4] reports a case of so-called **fetal or congenital rickets.** The mother was 48 years old, had borne 14 children, and was jaundiced at the birth of this child, which was well developed. At 2 weeks old it was admitted to the hospital with multiple fractures, occurring spontaneously; the cranial bones showed craniotabes and the ribs were soft, bending in with each inspiration and producing broad, shallow grooves on each side of the sternum; the long bones were not bowed nor enlarged at the ends. All of the fractures united, and at the age of 9 months the child was healthy, without signs of rickets. The author hesitates to classify the case in the absence of histologic examination of the bones. J. Cantlie[5] reports a case in a girl 13 years old, describing the condition and referring to the rarity with which these patients survive to adult life. F. C. Abbott[6] reports what he holds to be the most conclusive case of congenital rickets yet published. The infant died at the age of 14 months, showing then a large cranium, no teeth, rachitic rosary, spinal curvature, a deep longitudinal groove on each side of the sternum, and curvatures of the clavicles and long bones of the extremities, as in rachitis. The latter deformities were the natural curves which the bones assume in the intrauterine position, and the author attributes them to intrauterine pressure, the child being one of twins. There was a possibility of syphilis. Sections of the bones showed the characteristic changes of rachitis.

Rachitis.—R. Fischl[7] gives an extensive consideration of the various theories and factors commonly advanced to explain the **etiology of rachitis.** In a convincing way he shows that none of them approach at all near to the true etiology, pointing out the fallacies of all, from the geographic theory and that of social status through the theories of artificial feeding, of lessened absorption or increased elimination of lime, and all the other theories to that of infection. He believes that Stoeltzner and Kassowitz are on the right track in seeking the solution in the realms of cellular pathology and biology.

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[1] Jahrb. f. Kinderheilk., vol. LV, 1902.
[2] Jahrb. f. Kinderheilk., vol. LIV, 1901.
[3] Ref. in Jahrb. f. Kinderheilk., vol. LV, 1902, p. 357.
[4] Lancet, Aug. 17, 1901.
[5] Polyclinic, vol. VI, No. 3
[6] Brit. Med. Jour., Sept. 7, 1901.
[7] Arch. f. Kinderheilk., vol. XXXI

W. Stoeltzner[1] has used **suprarenal extract** tablets in additional cases of rachitis, and concludes that the Rachitol tablets are without influence, but that the English tablets did good, but were of varying effect. He does not consider it a valuable addition to the therapy, as the results can be obtained as easily in other ways.

Leo[2] reports 2 cases of rachitis treated with **phosphorus,** dying with toxic symptoms. The dose in each case had been 1 mgm. daily, one child taking it for only 15 days, the other for 6 months. Widespread fatty degeneration was found. T. Hryntschak[3] gives a rather sarcastic criticism of the treatment of rachitis by phosphorus, as urged by Kassowitz.[4] Hryntschak claims that long before the last dose of the phosphorus and cod-liver oil preparation is taken the phosphorus has nearly if not quite disappeared, that the mixture disturbs the digestion, and that there are no physiologic grounds for the administration of the drug, as the results are not so striking but that they may be attributed to the natural evolution of the disease. The author quotes Zweifel[5] and Monti[6] as holding the same opinion. A. Heiduschka[7] reviews Zweifel's publication and goes over the chemistry of phosphorus and cod-liver oil, finding that, as dispensed at various drug-stores in Leipzig, the reduction in the amount of phosphorus is so slight that it may be practically disregarded.

Scurvy.—v. Starck[8] in remarks on Möller-Barlow's disease, agrees with the American belief that it is infantile scorbutus and has no special relation to rickets or hereditary syphilis. He refers to its relatively greater frequency in certain districts like Hamburg, assigns **monotony of diet** to an important place in the etiology, and finds that it is difficult to say in advance in a given case what diet will agree.

F. Huber[9] reports a case of scurvy in a boy 13 months old weighing only 8 pounds and 1 ounce on admission and having almost universal **subcutaneous ecchymoses.** Rapid improvement followed the treatment, which consisted of orange juice, whisky, water by mouth, and salt solution by rectum to fill the tissues, boiled milk and barley-water (1 to 4) in small amounts, increased gradually, the child refusing to take fresh milk; later mutton broth, soft-boiled egg, and scraped meat were given, the child gaining 5 pounds in 2 weeks (Figs. 1 and 2).

J. McCaw[10] reports an atypical case in an infant 10 months old fed on **condensed milk and proprietary foods.** The symptoms were fever and the passage of blood by mouth and rectum. Appropriate treatment was followed by recovery in 8 days.

G. Carpenter[11] reports a case in a boy with rachitis 5½ years old. E. L. Pierson[12] also reports one. In reporting a case in an infant 11 months old, fed on Gärtner's humanized milk, J. Comby[1] mentions 8 similar cases out of 12 instances of scurvy, and deprecates the use of preserved milks, especially modified and laboratory milks [using these terms with different meanings from those employed in America].

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[1] Ref. in Arch. f. Kinderheilk., vol. xxx.

[2] Jahrb. f. Kinderheilk., vol. LIV, 1901.

[3] Arch. f. Kinderheilk., vol. XXXII.

[4] Wien. med. Presse, 1901, No. 1.

[5] Wien. klin. Woch., 1901, No. 2.

[6] Ibid., No. 3.

[7] Arch. f. Kinderheilk., vol. XXXIII.

[8] Münch. med. Woch., No. 23, 1901.

[9] Arch. of Ped., Nov., 1901.

[10] Brit. Med. Jour., No. 2131.

[11] Lancet, May 3, 1902.

[12] Boston M. and S. Jour., Oct., 1901.

Diabetes.—A. Fisch[2] reports a case of diabetes in an infant 11 months old, following acute gastroenteritis. Thirst was marked and emaciation progressed rapidly, with a fatal termination in 18 days. A. C. Cotton[3] reports a case in a girl 6½ years old, the disease lasting not quite a year, when coma developed. H. Stern[4] reports a case in a girl 4½ years old, with marked improvement following a rigid antidiabetic diet and antipyrin and codein medicinally. The author refers to the rarity of the disease under 5 years of age [*vide* Year-Book for 1902, p. 338], its brief duration, and the varying frequency of sex before and after 5 years, boys being in a majority in the younger group and girls in the older. The disease may

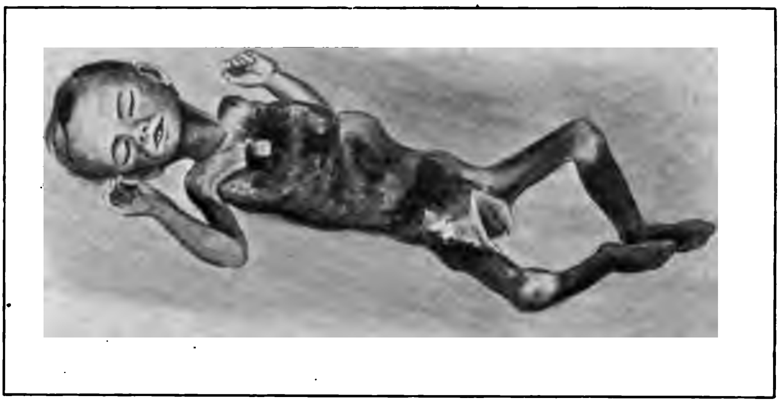

Fig. 1.—Huber's case of infantile scurvy and marasmus. Condition of the infant on admission to the hospital (Arch. of Ped., Nov., 1901).

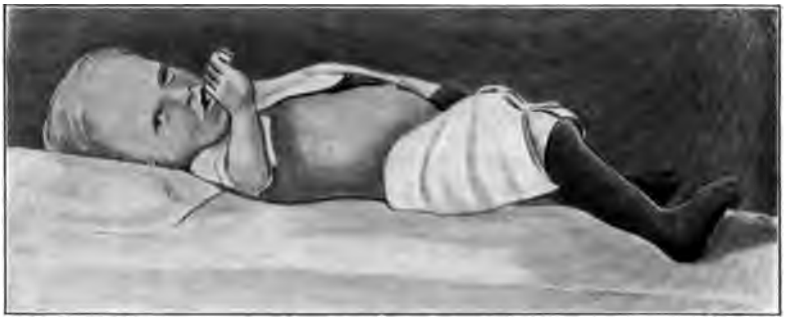

Fig. 2.—Huber's case of infantile scurvy and marasmus. Showing the improvement in the infant's condition (Arch. of Ped., Nov., 1901).

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[1] Arch. de Méd. des Enf., Aug., 1901.
[2] Pester med.-chir. Presse, 1901.
[3] Jour. Am. Med. Assoc., Sept. 7, 1901.
[4] Arch. of Ped., June, 1902.

be latent for some time, and is often preceded by a febrile attack or by one of the acute infections.

G. Finizio[1] reports on 14 cases of **alimentary glycosuria,** the patients being between 6 months and 6 years of age.

Lithemia.—J. Comby[2] gives a systematic discussion of the various manifestations of lithemia in childhood, which are common but are not always recognized as gouty. After heredity is mentioned, the pathogenesis is obscure and the treatment mainly dietetic and hygienic.

Rheumatoid Arthritis.—Moncorvo[3] collected 48 cases of rheumatoid arthritis in childhood, and reports one in an infant, the first signs having been noticed at 2 months of age.

DISEASES OF THE DUCTLESS GLANDS.

Cretinism.—H. Koplik and J. Lichtenstein[4] refer to stigmas of degeneration as deviations from the normal physical conformation and occurring in individuals morally and mentally deficient. These stigmas rarely affect vital structures, and many of them are often seen in normal persons. There are some which are pathognomonic of degeneration. To the latter class the authors add a stigma observed especially in cretins, consisting of a prominence near the antithenar eminence and over the pisiform bone; the sign was present in all of the cretins studied, and was also found in 2 cases of microcephalic idiocy, in a hydrocephalic idiot, and in a dwarf of marked mental degeneracy with goiter. It was not present in a number of Mongolian idiots, but it was found in 2 intelligent children who had macroglossia with polydactylia.

C. S. Millet[5] reports 2 cases of cretinism improving under treatment; the author finds that scarcely 100 cases have been reported in North America. Bourneville and Laurens[6] report a case coming under treatment at 16½ years and improving steadily in 2½ years. F. A. Packard and Alfred Hand, Jr.,[7] report a case in a boy 5 years old, with all the typical manifestations. Improvement was rapid under thyroid treatment, but a severe typhoid infection set in and ran a rapidly fatal course. At the autopsy the thyroid gland was atrophic, with overgrowth of connective tissue and calcareous degeneration of the blood-vessels. The authors query whether the thyroid treatment lessens resistance to the acute infections, a number of cretins having died of measles or some other infection while taking the remedy. Bezy and Stoianoff[8] report a case in a girl 5 years old, with a cure following rapidly increasing doses of the thyroid extract up to 24 grains a day.

Thymus.—R. Cruchet[9] gives a careful consideration of the thymus gland in childhood, its macroscopic appearance and anatomic relations. Taillens[10] reports 2 cases of thymus death and states that the fact of

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[1] La Pediatria, July, 1901.
[2] Arch. de Méd. des Enf., vol. v, Nos. 1 and 2.
[3] Rev. mens. des Mal. de l'Enf., July, 1901.
[4] Arch. of Ped., Feb., 1902.
[5] Boston M. and S. Jour., No. 15, 1901.
[6] Progrès méd., No. 23, 1901.
[7] Am. Jour. Med. Sci., Sept., 1901.
[8] Presse méd., Aug. 10, 1901.
[9] Rev. mens. des Mal. de l'Enf., Sept., 1901.
[10] Rev. méd. de la Suisse rom., June 20, 1901.

sudden death from hypertrophy of the thymus cannot be disputed. The mechanism is either toxic on the heart, or mechanical by pressure on the trachea, the former being very rapid and not subject to treatment, the latter slower and relieved only by operation.

Exophthalmic Goiter.—Wautski[1] reports a case of Basedow's disease in a boy 5 years old, all of the characteristic symptoms being present; improvement has followed the use of arsenic and galvanism. M. G. Variot[2] reports a case in a boy 4½ years old with the typical symptoms of exophthalmos, goiter and tachycardia. It followed pertussis complicated by bronchopneumonia. Soda salicylate was used without benefit, but thyroid extract and tonics caused great improvement in 2 months.

DISEASES OF THE GENITOURINARY SYSTEM.

Lop[3] reports **precocious menstruation** in a girl of 6 years, 5 regular monthly flows having occurred, the general health being good.

F. S. Churchill[4] reports a case of **cyclic albuminuria** in a girl 11 years old. There was a family history of cardiovascular disease and a personal history of measles, tonsillitis, and some intestinal infection of 12 days' duration. During the tonsillitis albuminuria was noticed, and this did not disappear subsequently; it was soon observed that its presence was cyclic, the urine voided before 10 A. M. being free and all passed between 10 A. M. and 8 P. M. containing albumin in varying but considerable amount. Examination of the sediment showed casts, red blood-cells, and frequently calcic oxalate crystals. The author reviews the literature, giving the theories as to the etiology and emphasizing the importance of following up such cases and keeping them under observation. H. Gillet[5] discusses the condition and analyzes 15 cases in children. H. Méry[6] handles the subject very thoroughly, the article being too extensive for an abstract. Porge[7] points out some of the distinctions of the different types—the orthostatic, the cyclic, and the digestive. He finds in explanation that the theories of nervous action, of dystrophy, and of digestion are insufficient to explain the etiology, which in most cases can be accounted for by the mechanical theory; thus, nearly every case shows cardiac, circulatory, and renal disturbances (palpitation, dilation of right auricle, lowered arterial tension), and diurnal oliguria, with a lessened excretion of the solids, as compared with the nocturnal urine.

Simon and Maheu[8] report a case of **hemoglobinuria** in a choreic girl, 9 years old, to whom 9 grams of antipyrin were given in 5 days.

T. Luzzatti and P. Sorgente[9] studied carefully a patient with **paroxysmal hemoglobinuria,** the attacks occurring frequently in winter or on exposure to cold (air or water). As a result of their observations they found the hemoglobinuria to depend on a hemoglobinemia, and this in turn was due to a hemolytic action of the blood-serum, brought about by a combination of cold with stasis of the blood-current. The theories to explain why cold has this action are reviewed, but the authors could not confirm any of them by animal experimentation.

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[1] Med. Woch., No. 32, 1901.
[2] Gaz. des Mal. Inf., vol. IV, No. 3.
[3] Gaz. des Hôp., Sept. 12, 1901.
[4] Arch. of Ped., Sept., 1901.
[5] Arch. de Méd. des Enf., June, 1901.
[6] Rev. mens. des Mal. de l'Enf., Nov., 1901.
[7] Id., Dec., 1901.
[8] Ibid.
[9] Arch. f. Kinderheilk., vol. XXXII.

W. P. Herringham[1] reports a case of **dropsy without albuminuria** ending fatally. He reviews the theories advanced concerning the condition, and suggests the term essential toxemic dropsy. [The absence of albuminuria does not exclude nephritis, as casts and renal epithelium occur not rarely without albuminuria, so a case ought not to be placed in the author's category without first having microscopic examination of the centrifuged sediment from a fresh specimen of urine.]

S. Silvestri[2] studied **Ehrlich's diazo reaction** in children's urine and found it entirely negative in 90 healthy children. In typhoid fever it was invariably positive, corresponding in intensity to the severity of the disease and appearing on the fourth or fifth days. In measles it was always present at the acme, but the intensity and severity did not always correspond; twice after disappearing it returned with the occurrence of a pulmonary complication. In 3 cases of German measles the reaction was absent. Two cases of pulmonary and 5 of miliary tuberculosis and 3 out of 5 cases of tuberculous meningitis gave positive reactions. Depending on the severity of the disease, the reaction was present in from 25 % to 40 % of the cases of croupous pneumonia, diphtheria, scarlet fever, erysipelas, and pyemic processes, while influenza gave only 20 % of positive results, only the severe or complicated cases reacting. Entirely negative results were obtained in pertussis, rickets, hereditary syphilis, anemia, eclampsia, catarrhal bronchitis and pneumonia, acute febrile gastroenteritis, follicular enteritis, acute and chronic peritonitis.

Spolverini[3] discusses the **acute infectious and toxic nephrites** of childhood. He found in 3 cases of scarlet fever streptococci in pure culture in the urine; the urine of toxic nephritis gave no growth. Bacterial nephritis is usually glomerular, while toxic nephritis is usually tubular, but mixed forms may occur. The bacterial or septicemic forms are characterized by intermittent fever, and the urine of a post-scarlatinal nephritis is infectious. In 150 cases of measles true post-morbillar nephritis developed 6 times, and in 180 cases of diphtheria but 5 developed nephritis (a proof of the good action of the antitoxin in this direction).

I. Democh[4] places on record the third reported case of contracted kidney in infancy. The patient, a boy 2 months old, had convulsions, cardiac hypertrophy, anasarca, and pneumonia, the autopsy showing granular kidneys, slightly cystic. The author attributed the condition to a parenchymatous nephritis dependent on gastroenteritis, and did not consider it to be congenital.

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[1] Brit. Med. Jour., No. 2123.

[2] La Pediatria, May, 1901; Arch. of Ped., Sept., 1901.

[3] Ann. de méd. et de chir. inf., 1901.

[4] Arch. f. Kinderheilk., vol. XXXIII.

A. Primrose[1] gives the notes of a case of nephritis of 6 months' standing in a boy 10 years old, with marked anasarca. An **operation was done,** cutting down to the right kidney, which was enlarged and ashy-purple, and an incision was made along the convex border 1½ inches long and ¼ inch deep, drainage being left in. Improvement followed for 3 weeks, but then the ascites returned, necessitating tapping, so an operation was done on the left kidney, stripping it of its capsule completely. Pneumonia developed 1 week later with recovery. Two months after the last operation there was but a trace of albumin with very few casts, and the boy was gaining in strength. [It is to be supposed that medicinal means were exhausted before resorting to operation.]

W. Jepson[2] reports a case of **unilateral congenital cystic kidney** removed under chloroform when the child was 14 months old, the operation lasting but 14 minutes. The child made a complete recovery. J. T. Shirlaw[3] reports a similar case of such size as to cause dystocia. Operation was done when the child was 9 weeks old, and 700 cc. of fluid were evacuated, the child recovering.

Romniceanu and Robin[4] have studied during 14 years 150 cases of **vaginitis** in girls from 2 to 13 years old, gonococci being present in 130. Many complications were observed, and relapses occurred as late as 7 years after apparent cure. Modes of infection were various, complicating the medicolegal aspect. For treatment, boric acid, 4 %, and silver nitrate, 1 % to 2 %, are recommended.

J. Langer[5] discusses the value of the acetic acid and potassium ferrocyanid test for albumin as applied to urine from diphtheria patients. On adding the acid, a turbidity often appears which is not always albumin, but may be urates which are thrown down by the acid because of the concentration of the urine. The author lays stress on the proper estimation of the albumin, because in private practice doctors do not usually examine the sediment. [We suppose that this is unfortunately so, although inexcusable.]

J. L. Morse[6] discusses the renal complications of the acute diarrheas of infancy, pointing out the complex etiology, the obscure symptomatology, and the possibility of complete recovery.

J. Poscharynski[7] found in samples of urines from 14 patients acid-proof bacilli, care being taken to exclude the smegma bacilli. The significance of the finding is inexplicable at present.

H. Roeder[8] reviews the subject of the **determination of the freezing-point of blood and urine,** its present position and its general significance in the question of renal insufficiency.

[1] Canad. Jour. of M. and S., vol. XI, No. 3.
[2] Jour. Am. Med. Assoc., Sept. 28, 1901.
[3] Brit. Med. Jour., Nov. 16, 1901.
[4] Wien. med. Presse, No. 43, 1901.
[5] Jahrb. f. Kinderheilk., vol. LV, 1901.
[6] Med. News, June 7, 1902.
[7] Arch. f. Kinderheilk., vol. XXXII.
[8] Arch. f. Kinderheilk., vol. XXXIV.

DISEASES OF THE NERVOUS SYSTEM.

C. Cattaneo[1] gives a condensed report of an extensive study of the **reflexes in infancy,** one of his conclusions being that, on account of their constancy, the only reflexes which can have any pathologic significance are the plantar and the patellar.

Feeble-mindedness in children is to be separated, F. Warner[2] states, from mere mental dulness or ignorance. W. L. Andriezen[3] refers to the difficulty of diagnosis in infants under 12 months of age, the chief manifestations being the lack of variety and spontaneity in the child's movements and inability to imitate. E. B. Angell[4] points out that a single anatomic stigma is of little importance, but that the association of anatomic and physiologic stigmas is very significant. A. W. Wilmarth[5] discusses the diagnosis of **backwardness in children,** showing that the line between the normal and the backward child is not a sharp one. The mental points for observation are acuteness of perception, power of attention, strength of memory, accuracy of reasoning and judgment, and growth of normal sense. When the mental growth is delayed, the recognition of backwardness is aided by certain physical defects, among which are disturbances of articulation not due to mechanical faults or deafness; defects in coordination or awkward gait; defective physical growth and lack of acuteness in the special sense-organs. G. H. Makuen[6] points out the value of studying the speech of backward children with reference to the diagnosis and prognosis. The cause of backwardness is not rarely referable to some abnormity of structure in the peripheral organs, especially those of speech, upon which arrested cerebral development is dependent. As a prognostic point, the extent to which speech-development occurs in backward children will indicate the possibility of general development.

A. Castex[7] reviews the **etiology of deaf-mutism,** and finds that it is as often congenital as acquired. In the latter group important causes are: (1) consanguinity of the parents; (2) tuberculosis, syphilis, alcoholism, or lead-poisoning in the parents; (3) accidents of pregnancy.

Gutzmann[8] advises careful regulation of the diet of stuttering children, who are usually nervous or neurasthenic, and as a rule are on a rich meat diet; this should be limited, and vegetables substituted, with avoidance of alcohol, with the use of cold baths and exercise in the open air.

A. B. Marfan[9] reports without comment the sad **history of a family whose father became addicted to the use of cocain** following an operation in the nasal passages. The first child, 13 years old, is healthy; the second, 8 years old, conceived 2 months after the beginning of the cocain habit, is intelligent and nervous; the third, conceived at the height of the cocainism, is a hopeless idiot; and the fourth, 10 months old, is a microcephalic idiot.

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[1] Jahrb. f. Kinderheilk., vol. LV, 1902.

[2] Brit. Med. Jour., Oct. 26, 1901.

[3] Ibid.

[4] Ibid.

[5] Jour. Am. Med. Assoc., Oct. 12, 1901.

[6] Jour. Am. Med. Assoc., vol. XXXVII, No. 15.

[7] Arch. de Méd. des Enf., Sept., 1901.

[8] Ref. in Arch. f. Kinderheilk., vol. XXXIII.

[9] Rev. mens. des Mal. de l'Enf., Sept., 1901.

P. F. Barbour[1] reports a case of **Mongolian idiocy** in a negro boy, 11 years old. Thyroid extract was given, the only change noticed being a somewhat rapid growth. J. Park West[2] refers to Telford Smith's communication on the curve of the little finger existing in Mongolian idiots, which curve is due to a shortening of the second phalanx and a lateral displacement of the distal phalanx. West studied the fingers of 605 normal children and 9 Mongolian idiots, and found that the 3 types of straight, moderately curved, and markedly curved little fingers were present in both groups.

C. Stamm[3] reports briefly 8 cases of **nodding spasm** and discusses the condition, attributing to dark dwellings an influence only in so far as they contribute to a rachitis which is always present, the nodding being looked on as a symptom of rachitis resulting from an increased vascularity of the inner cranial periosteum and of the meninges. As a further proof of its rachitic origin, he points to the specific action of phosphorus, the use of which was followed by diminution of the movements in 8 days and their disappearance in a few weeks. [We agree with the author that rachitis is present in all of these cases, the nutrition being always disturbed; we must deny that phosphorus is the only specific, for we have seen a nearly equal number of cases recover in a few weeks without medication and with only careful attention to the diet.] S. Amberg[4] reports a case in a male infant 5 months old, unusual in that the head-movements persisted during sleep, and when the child was laid on his back, when awake, there were twitching movements of the arms and legs of both sides.

R. A. Peters[5] discusses the diagnosis of **hysteria in childhood,** referring to its difficulties and to his results in treatment by hypnosis and other forms of suggestion. Paresis and enuresis dependent on hysteria were the conditions most benefited. M. Thiemich[6] believes that enuresis depends on hysteria entirely, and not on muscular weakness or local causes, and he therefore recommends change of surroundings, isolation of the patient, faradization, and general hygiene. The success of other methods he attributes to suggestion.

D. Crisafi[7] makes the distinction between true **pavor nocturnus** and the symptomatic forms. The latter depend on some source of reflex irritation, and are more properly termed pavoriform. The true attacks last from ½ to 2 hours and are characterized by terrifying hallucinations. Urine is voided at the close of the attack and the child usually has no recollection of the attack.

A. A. Eshner[8] reports a case of nodding spasm, discussing its relation to **spasmodic torticollis,** which is observed almost exclusively in adults; he appends the notes of an illustrative case.

A. Moussous[9] treats of the differential diagnosis of a form of chorea termed by him **chorea of degeneracy,** from the choreas known by the names of Sydenham, Huntington, and Gilles de la Tourette. The author's form often begins, like ordinary chorea, suddenly, but it may persist from childhood on into adult life. The movements are to a small extent controlled by voluntary motion, so that the patients do not become helpless. The mental state comprises usually lack of ability to fix the attention, an emotional and changeable character with irascibility. Physical signs of degeneracy are ordinarily present.

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[1] Arch. of Ped., April, 1902.
[2] Arch. of Ped., Dec., 1901.
[3] Arch. f. Kinderheilk., vol. XXXII.
[4] Arch. of Ped., Nov., 1901.
[5] Vratch, June 2, 1901.
[6] Berl. klin. Woch., July 22, 1901.
[7] La Pediatria, vol. IX, No. 10.
[8] Arch. of Ped., Aug., 1901.
[9] Rev. mens. des Mal. de l'Enf., Nov., 1901.

T. Froelich[1] discusses the **etiology of chorea minor,** showing that while many writers refer to the relationship between chorea and rheumatism, others deny it; his own figures showed preceding or contemporaneous rheumatism in 31.9 %, other infectious diseases in 8.9 %, unknown infections in 34 %, and no preceding infection in 25.5 %. G. Singer[2] has found in 5 cases of acute rheumatism and 2 of chorea a general infection with various cocci, which he views as causal. O. Heubner[3] states that the close etiologic **relationship between chorea and rheumatism** is proved by the fact that many choreics (40 % in his 77 cases) have had articular rheumatism, many of them belong to rheumatic families, both diseases show the so-called rheumatic exanthems. Looking on chorea, then, as an equivalent of a disease marked by a tendency to inflammation of the heart, the author therefore advises rest in bed for a long time. He also gives arsenic and a sweating pack every few days.

S. Laache[4] analyzes 40 cases of chorea. He looks on the disease as a **neurosis and not an infection,** explaining those cases with demonstrable postmortem lesions of endocarditis, etc., as instances of secondary infection. L. Fornaca[5] practised lumbar puncture in 3 cases of chorea, obtaining streptococci from the fluid in one case following erysipelas; benefit seemed to follow the procedure. R. S. Farreras,[5] after ordinary serum had failed, used with success injections of bovine physiologic serum, 10 cc. every 2 days, in chorea in a boy 9 years old, recovery following rapidly.

H. Thomas[6] reports a case of chorea in a girl 16 years old, there suddenly occurring loss of vision in the left eye due to embolism of the central artery of the retina. Eye-complications in chorea are rare, and the author found only 6 other cases, all of which the reporters advanced as upholding the theory of **embolism as causing chorea,** but Thomas does not agree with this, thinking that the lesion, if found, will be on the afferent rather than on the efferent paths. H. N. Moyer[7] discusses the psychoses of chorea.

A. C. Cotton[8] reports a case of **amaurotic family idiocy** in a Hebrew child aged 2 years. He emphasizes the difficulty of diagnosis until the eyeground is examined. H. Higier[9] reports 3 cases, 2 occurring in one family; the children developed normally for 7 or 8 months and then the characteristic symptoms of idiocy, blindness, and weakness would appear, followed by death. Both of the families were Jewish, and the author refers to the fact that almost all of the cases reported have been in Jewish families. Falkenheim[1] reports 4 cases and reviews the literature with abstracts of all the cases published; the article is illustrated with a lithograph of the characteristic eyeground (Fig. 3).

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[1] Jahrb. f. Kinderheilk., vol. LIV.

[2] Wien. klin. Woch., No. 20, 1901.

[3] Ref. in Arch. f. Kinderheilk., vol. xxx.

[4] Norsk. Mag. f. Laeg., 1901.

[5] La Rif. Med.; Arch. of Ped., Aug., 1901.

[5] Arch. de Gin., Obstet., y Ped., Ano xv, No. 2.

[6] Johns Hopkins Hosp. Bull., vol XII, No. 127.

[7] Jour. Am. Med. Assoc., Nov. 23, 1901.

[8] Arch. of Ped., Jan., 1902.

[9] Neurolog. Centralbl., Sept. 16, 1901.

Biro[2] reports 5 cases of **Friedreich's ataxia,** 3 of them in brothers aged 2, 7, and 15 years; the differential diagnosis is considered. E. Weber[3] reports 3 cases in one family, there being 2 other healthy children; the disease began in each case at the age of 6 years.

J. T. G. Brooksbank[4] reports 3 cases of **tabes dorsalis occurring in children congenitally syphilitic,** the differential diagnosis from Friedreich's hereditary ataxia resting on the evidences of syphilis and the presence of the Argyll-Robertson pupil. Anesthesia and incoordination have not developed.

G. Carriere[5] reports a case of **paramyoclonus multiplex** in a boy 10 years old, of neuropathic stock. Attacks of pain with involuntary movements of the trunk and extremities occur frequently, lessening when attention is drawn to a member or voluntary movement is made. The author has seen 3 of the 32 cases reported, and believes that the basis is hysteria, suggestive treatment being indicated.

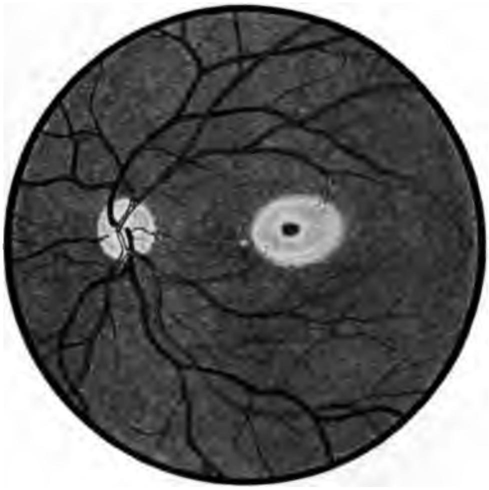

Fig. 3.—Characteristic eyeground in amaurotic family idiocy (Falkenheim, in Jahrb. f. Kinderheilk., vol. LIV).

G. Alsberg[6] reports 4 cases of **porencephaly** and discusses at length the theories of causation. Henoch believes that while it may be a fault of development, it is usually caused by an inflammatory process, with a more or less hemorrhagic effusion, absorption taking place with cyst formation. Heschl attributes it to a fetal meningitis with subsequent hydrocephalus. Kundrat's view that it is the result of a mechanical disturbance of the circulation in the region supplied by the artery of the sylvian fissure is made probable by the fact that porencephaly always appears in this situation. The theory of traumatism has little to support it. The author does not limit porencephaly to a congenital condition, but states that it may be acquired, the distinction between the two being made

[1] Jahrb. f. Kinderheilk., vol. LIV

[2] Deut. Zeitschr. f. Nervenh., Heft 2.

[3] Deut. med. Woch., Sept. 26, 1901.

[4] Lancet, No. 4087.

[5] Presse méd., Aug. 7, 1901.

[6] Arch. f. Kinderheilk., vol. XXXIII.

by the arachnoid membrane which lines the cavity in the congenital form. Of the author's cases the condition was bilateral in 2; external hydrocephalus was present in 1 and there was descending degeneration of the spinal cord in the fourth; 3 had cerebral symptoms, one of them showing marked opisthotonus; one patient who died of croup had shown no symptoms of central lesion, but at the autopsy there was found a cyst in the left hemisphere containing 200 cc. of hemorrhagic fluid. F. von Szontagh[1] reports a case of congenital bilateral porencephaly in an infant living only 23 hours. The author considers the case to support the view that porencephaly is the result of failure of development.

Rolly[2] reports 4 cases of **congenital bilateral spasticity,** which he groups under the term Little's disease. The autopsies showed overgrowth of connective tissue in the central nervous system and bloodvessels. As paresis was not present, the author thinks 2 clinical groups of Little's disease should be made, 1 with paralysis and the other without, and that for the latter group the anatomic basis is a sclerosis of the central nervous system. Zappert,[3] in reviewing the articles, points out that as the fathers of all the children had had syphilis, the lesions might only be those of hereditary infection, and that further observations are necessary.

J. Kollaritz[4] reports a case of **acute bulbar palsy** in a child and refers to the literature in which similar cases are reported, the acute poliomyelitis affecting the centers of the cranial nerves.

J. Grósz[5] reports a case of functional bulbar palsy (Oppenheim's bulbar palsy without anatomic lesion; Strümpell's asthenic bulbar palsy) in a girl who at the height of the disease could not speak. There was inability to swallow except when lying down, and then only liquids would go down. Incoordination of the arms and legs, tremor of the tongue and eyelids, exaggeration of the patellar and abdominal reflexes, and a normal condition of the senses were present. With bromid and iodid recovery followed in 2 weeks. The author states that hysteria could not be excluded.

P. Fabris[6] studied 22 cases in an epidemic of **acute infantile palsy** and gives as elements in the etiology a neuropathic tendency, frequently inherited; certain meteorologic influences; and a specific germ which elaborates its poison in the body at a distance remote from the cord, upon which the poison has a selective action.

W. Nissen[7] details the symptoms in 5 cases of **brain-tumor,** the autopsies in 3 showing cheesy tumors of the corpora quadrigemina, the diagnosis in the others being clinical, the same region being probably affected.

O. Heubner[8] reports a case of **multiple gliomas** of the cord and internal hydrocephalus, in a girl 7 years old.

R. Millon[9] reports 2 cases of traumatic zona, which consists of an eruption of **zoster** in the nerve-distribution of a part, following a blow.

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[1] Pest. med.-chir. Presse, 1901.

[2] Deut. Zeitschr. f. Nervenheilk., vol. XX.

[3] Jahrb. f. Kinderheilk., vol. LV, 1902.

[4] Id., vol. LIV.

[5] Arch. f. Kinderheilk., vol. XXXIV.

[6] La Pediatria, vol. IX, No. 5.

[7] Jahrb. f. Kinderheilk., vol. LIV, 1901.

[8] Arch. f. Psych. u. Nervenheilk., vol. XXXIV.

[9] Arch. de Méd. des Enf., July, 1901.

Bourneville and Crouzon[1] report a familial disease in 2 boys, the clinical diagnosis being **spastic infantile diplegia and idiocy,** the autopsies showing atrophy of the cerebellum and pons, with lessening of the fibers of the pyramidal tracts in the medulla.

W. Ewart and W. L. Dickinson[2] treated 2 cases of **chronic hydrocephalus** by tapping and then introducing sterile air in order to relieve the brain of the pressure and weight of fluid. They state that the operation may be repeated without harm, but that in some cases a single operation may suffice. They caution against sudden movements of the head or the eliciting of the succussion splash as long as the hydropneumencephalus persists. V. Immerwol[3] gives the results of treatment in 10 cases of hydrocephalus; 9 were congenital and 1 was acquired. Of the former, all received antisyphilitic treatment, and in 5 the lateral ventricle was tapped, iodin being injected in 1, the 4 others having lumbar puncture. Of these 9, but 1 was cured. The acquired case was cured by repeated lumbar punctures and the internal use of sodium iodid. The author recommends repeated lumbar puncture in all cases of acquired hydrocephalus and in all the congenital cases also, which should receive, in addition, antisyphilitic treatment. Rocaz[4] reports a case of congenital hydrocephalus in a syphilitic infant, recovery following mixed treatment.

Ossipow[5] has made the effects of **lumbar puncture the subject of experimental study on dogs.** Two groups were used, the pressure being estimated in one group and then the fluid being allowed to flow by gravity; in the other group the fluid was aspirated. The operation was repeated a number of times on some of the dogs, and they were then killed and the central nervous system studied. Persistent hyperemia of the meninges and of the substance of the brain and cord followed removal of fluid. If the punctures were repeated, punctiform hemorrhages appeared in the upper lumbar and lower cervical regions and in some cases in the brain-substance. In the cases aspirated hemorrhage into the canal was not rare. Following the repeated punctures there were changes in the nerve-cells. The author therefore concludes that lumbar puncture is not a trifling operation [right], and that whenever the cerebrospinal fluid is drained there is danger of hemorrhage into the brain or cord [he should have added, to be logical, when done as he did it]; contraindications are therefore stated to be sclerosis or anemia of the cerebral vessels and lack of evidence of increased pressure in the cerebrospinal fluid. He states that the danger is not so great when only a small quantity is removed, and urges that the indications for the operation should be clear in each case and aspiration should not be practised. [All of these points have been well understood before this, the paper being of main value in showing the dangers of aspiration.] J. Abadie[6] refers to the great diagnostic value of lumbar puncture. The freezing-point in tuberculous meningitis is lower than normal. Bile-pigment has been found when excessive jaundice exists. The permeability of the pia-arachnoid may be tested by the administration of potassium iodid or methylene-blue by mouth or hypodermically and testing the fluid drawn subsequently.

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[1] Le Progrès méd., April 27, 1901.
[2] Brit. Med. Jour., Sept. 7, 1901.
[3] Arch. f. Kinderheilk., vol. xxxii.
[4] Jour. de Méd. de Bord., Sept. 22, 1901.
[5] Deut. Zeitschr. f. Nervenheilk., Heft 2.
[6] Jour. de Méd. de Bord., July 28, 1901.

DISEASES OF THE MUSCLES.

Kollarits[1] gives the pathology of a case of pseudohypertrophic muscular dystrophy, the patient being a boy 10 years old, unable to walk for 2 years and accidentally drowned while bathing. In addition to the usual changes in the muscles, there were found in the central nervous system dilation of the central canal, diminution of the fibers in Lissauer's zone, in the gray matter around the central canal, in the anterior and posterior commissures, and in Clark's column. The ganglion cells of the anterior cornua seemed small, but otherwise normal. The author believes that these changes were not secondary to the muscular degeneration but show an impaired development similar to that in the muscles.

A. A. Eshner[2] reports 2 cases of **progressive muscular dystrophy** in brother and sister, the disease beginning in the boy at about the fifth year and in the girl at about the seventh year; the latter is now 32 years old and almost entirely helpless. B. Sachs and H. Brooks[3] report a case under observation from 13 years of age until death 11 years later. A younger brother presented Erb's juvenile type of the disease, and another brother had the ordinary form of pseudohypertrophy. Symptoms first developed in the patient at 10 years of age, when he broke his leg, after which he could not walk well; after typhoid fever at 12 he could not walk at all, and he ultimately became completely helpless, unable to raise his head or to move his limbs. The autopsy showed fibrosis of all the skeletal muscles, without lesions of the central nervous system except degeneration of a few cells of the posterior ganglions, probably due to the terminal infection. The findings confirm the view that the disease is a primary muscular condition. The authors have had marked improvement in 2 cases following active exercise.

C. F. Gardiner[4] reports a case of **congenital myotonia** in a boy of 6 years, whose mother was troubled during pregnancy with extreme slowness of muscular action, which disappeared after delivery. The author inclines to von Bechterew's theory that the disease is a disturbance of metabolism.

TUMORS.

E. Bonnaire and Decloux[5] review the literature of **congenital lymphadenoma in the newborn** and report a case in a male infant 5 months old with an abdominal tumor thought to be a hypertrophied syphilitic spleen. At the autopsy this was found to be a lymphadenoma of the left suprarenal gland with similar deposits in nearly all the organs. A. S. Warthin[1] contributes an important report of a similar case in an infant 4 days old, who had suppression of urine for 24 hours and died in convulsions. The pathologic diagnosis was: **adenoma of both adrenals** (arising in the fascicular zone); retrogressive changes in the adrenals of Marchand (small collections of adrenal tissue along spermatic vessels or broad ligament, which the author considers are probably normal); acute degenerative nephritis; fatty degeneration and cloudy swelling of liver; congestion of liver, lungs, spleen, and kidneys; pressure atrophy of left kidney; anomaly of left lung (3 lobes). The degenerative changes in the kidneys and liver were probably toxic from the growth. The author gives a thorough report and points out that the appearance of the larger growth (in the left suprarenal) might have led to a hasty diagnosis of hemorrhage, although characteristic of adrenal adenoma, a capsule of living adrenal tissue surrounding a degenerated semifluid brownish substance.

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[1] Deut. Arch. f. klin. Med. vol. LXX.
[2] Phila. Med. Jour., June 7, 1902.
[3] Am. Jour. Med. Sci., vol. CXXII, No. 1.
[4] Arch. of Ped., Dec., 1901.
[5] Presse méd., July 13, 1901.

Josias and Tollemer[2] report a case of **generalized malignant lymphoma** in a child 9 years old, starting from the left tonsil and following an attack of streptococcus angina; tracheotomy was necessary to relieve the dyspnea caused by the growth of the glands, the tonsil filling the mouth. Microscopically the lymph-glands showed the structure of a lymphosarcoma and lymphoid infiltrations were found in the liver, spleen, marrow, and kidneys. There was no leukocytosis before death, but the differential count showed an increase in the proportion of the mononuclears at the expense of the polynuclears. [Inoculation experiments would have been interesting to see if the process could possibly have been tuberculous.]

P. Lereboullet[3] reports a case of **sarcoma of the pia mater** in a girl 4 years old, the symptoms resembling tuberculous meningitis. The growth, a spindle-celled sarcoma, started in the left auditory nerve with extension to the pia of the bulbo-protuberantial region and of the cord.

J. D. Malcolm[4] reports a case of abdominal tumor in a child 4½ years old, simulating tumor of the kidney. Operation showed 2 growths, one of the tail of the pancreas and the other metastatic in the gastrohepatic omentum, both being fibrosarcomas.

J. H. Jopson and C. Y. White[5] collect from the literature 21 cases of **sarcoma of the large intestine,** 6 being under 10 years of age. They report one in a boy 4 years old, the diagnosis during life resting between tuberculous peritonitis and tumor, autopsy showing a round-celled sarcoma with extensive metastasis.

L. Fischer[6] reports a case of **spindle-celled sarcoma of the thorax** in a boy 8 years old, the growth projecting forward from the chest-wall and evidently extending within the thorax, as the heart was displaced and there was great dyspnea.

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[1] Arch. of Ped., Nov., 1901

[2] Presse méd., No. 100, 1901.

[3] Id., No. 104.

[4] Lancet, No. 4096, 1902.

[5] Am. Jour. Med. Sci., vol. cxxii, No. 7.

[6] Arch. of Ped., May, 1902.

A. Schoenstadt[1] removed a sarcomatous kidney from an infant 6 months old who was in good health 7 months later.

J. Lorthioir[2] removed a **supposed uterine fibroma** from the vagina of a girl 3 years old. Recurrence took place in 2 months, when a total abdominal hysterectomy was done, the child recovering completely. The tumor proved to be a small round-celled sarcoma.

THERAPEUTICS AND TOXICOLOGY.

A. Baginsky[3] finds that the **indications for venesection in children** do not rest on any scientifically demonstrable blood-conditions, but on certain clinical phenomena of two kinds, one of mechanical disturbance of the circulation and the other of a toxic state. Whatever the cause may be which leads to dilation of the right heart with venous stasis, venesection is indicated when the following picture is present: small arteries with thready, rapid pulse, cyanosis, steadily increasing dyspnea, orthopnea with marked depression, restlessness with or without delirium, coldness and cyanosis of the extremities; suffocation with danger of immediate death; enlargement of the liver, albuminuria of high degree with the presence of morphotic elements, especially blood-cells. Whether these symptoms depend on an affection primarily cardiac, pulmonary, or renal makes no difference. With regard to the second indication for venesection, a toxic state, benefit is obtained only when the toxemia depends on the retention of the products of tissue-waste, as in uremia, in which bleeding often produces brilliant results. In the toxemias of the infectious diseases like diphtheria, bleeding has long been abandoned, and the absence of benefit after bleeding in such diseases, which was at once observed, is now confirmatory of Ehrlich's side-chain theory, as showing that the toxins are not circulating free but are in combination in the blood.

W. C. Hollopeter[4] describes the **technic of hypodermoclysis in pediatric practice,** which may be accomplished by the use of a large hypodermic syringe, the solution being at least of 115° F. and delivered at 106° F. into the tissues. The amount need not be large, 1 or 2 ounces often acting wonderfully well. Hypodermoclysis is of use in the anuria of the first 3 weeks of life, due to uric acid infarcts; in uremic coma following the infectious diseases; in shock caused by intestinal intoxication; in depression following any acute febrile disorder; in the atrophy of malnutrition; while in syphilis and tuberculosis it aids the other measures greatly.

L. E. Holt[5] reports a case of **gastrointestinal hemorrhage in a new-born infant,** controlled in 12 hours by suprarenal extract. One grain of the saccharated extract was given every hour for 6 doses and then lessened to half a grain, 12 grains in all being given. The child afterward thrived perfectly.

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[1] Berl. klin. Woch., No. 39, 1901.

[2] Jour. de Chir., vol. I, No. 8.

[3] Arch. f. Kinderheilk., vol. XXXI.

[4] Phila. Med. Jour., Dec. 7, 1901.

[5] Arch. of Ped., April, 1902.

F. W. E. Kinneir[1] reports the case of a boy, 10 years old, with a hemophilic family history, who fell and bit his tongue, **profuse hemorrhage** following. The wound was packed with powdered suprarenal extract and closed with collodion. Secondary hemorrhage occurred, and a fatal termination was prevented by the actual cautery, which stopped the bleeding.

D. B. Lees[2] exhibited a patient, a girl 8 years old, with **multiple neuritis** following the use of arsenic for chorea. The dose had been 15 minims of liquor arsenicalis, thrice daily for 5 weeks.

J. L. Morse[3] reports a case of arsenical poisoning in an infant 6 months old, the source having been the blue sateen lining of the basinette. The symptoms were occasional attacks of cyanosis with a systolic murmur, a progressive anemia with retardation of growth, and the passage of a reddish urine containing blood-cells and arsenic. Recovery was complete.

C. D. Slagle[4] reports a case of **morphin-poisoning** in an infant 4 weeks old, ¼ grain having been given by mistake; convulsions were absent, but cyanosis was present with a pulse of 20 and respirations of 4 per minute. The treatment consisted in strychnin, atropin, cutaneous stimulation, lavage of the stomach with strong coffee, and an enema of hot saline solution. Recovery followed. W. B. Burns[5] reports the case of an infant 4 weeks old to whom 12 drops of **chloranodyn** was given for intestinal pain in 2½ hours. Immediately after the last dose, convulsions set in and were followed by collapse, 12 hours of active treatment being necessary to restore the child. Reference is made to another instance in which $\frac{1}{32}$ grain of morphin in 2 doses 4 hours apart given to a month-old infant was followed by untoward results.

Lop[6] observed a boy in a marked state of collapse with cyanosis and abdominal cramps, this condition lasting for a day. The history was that he had drunk 2 glasses of iced lemonade half an hour before the attack, and had been wearing for several hours **shoes polished with a liquid dressing** which was found to **contain considerable arsenic.**

J. Gròsz[7] mentions several cases of **acute alcoholism** he has seen in children, convulsions being a marked feature of the intoxication. He refers to the pernicious custom [fortunately in America it is otherwise] of giving children alcoholic drinks almost daily "to strengthen them," a custom of the laity which has too often had its basis in the irrational prescribing of alcohol by the medical profession. [We wish that we could repeat our last comment at this point.] The author emphasizes the harmful effects exerted on the child's system by alcohol, and lays down as the sole indication for the administration of alcohol "those conditions in which we have to deal with a rapid loss of strength and suddenly appearing symptoms of heart-weakness." M. Kassowitz[8] reports a number of cases of acute and chronic alcoholism in children and discusses the value of alcohol medicinally, as a food, stimulant, stomachic, antipyretic tonic and stimulant in acute fevers, antiseptic, and a spur to the mental faculties, in all of which he opposes its use "openly, consistently, and decidedly." [At this writing we are observing a number of convalescents from typhoid fever to whom not a dose of alcohol was given, but we have not yet reached the point where we feel justified in withholding it in severe cases with weak pulses.]

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[1] Brit. Med. Jour., No. 2149. [2] Ref. in Arch. of Ped., Jan., 1902.
[3] Id., Sept., 1091. [4] Jour. Am. Med. Assoc., vol. xxxviii, No. 7.
[5] Memphis Med. Monthly, vol. xxi, No. 11.
[6] Arch. de Méd. des Enf., Jan., 1902. [7] Arch. f. Kinderheilk., vol. xxxiv.
[8] Jahrb. f. Kinderheilk., vol. liv, 1901.

F. W. Burton[1] reports a case of bromoform poisoning from the last dose in the bottle [as frequently happens].

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[1] Brit. Med. Jour., May 18, 1901.

PATHOLOGY AND BACTERIOLOGY.

By DAVID RIESMAN, M.D., AND A. O. J. KELLY, M.D.,

OF PHILADELPHIA.

TUBERCULOSIS.

Human and Bovine Tuberculosis.—During the past year the discussion relative to the identity and the intercommunicability of human and bovine tuberculosis has continued. Koch has reaffirmed his former position and has criticized adversely the work of some of those who contend for the identity and the intercommunicability of the two forms of the disease. Ravenel,[1] in an extremely valuable address, reviews the entire subject, and reaches the conclusion that the evidence at hand forces us to the opinion that human and bovine tuberculosis are but slightly different manifestations of one and the same disease, and that they are intercommunicable. Bovine tuberculosis, therefore, is a menace to human health. We are not in a position at present to define positively the extent of this danger, but that it exists cannot be denied. In the past there has probably been a tendency to exaggeration, but however great this may have been, it does not now justify any attempt at belittling the risk, and it is folly to blind ourselves to it. The eradication of bovine tuberculosis is amply justifiable from a purely economic standpoint; viewed in its bearing on human health it becomes a public duty. At the 1902 meeting of the British Medical Association a valuable discussion[2] on the relationship of human and bovine tuberculosis took place. Hamilton stated that it will be generally conceded that whereas the bovine bacillus excites, as a rule, a tuberculosis in cattle far more widespread and acute than that occasioned by the human organism, yet we are now in possession of evidence showing that the conclusions arrived at by Koch and Schütz are far too absolute. It has been proved beyond a shadow of doubt that the bacillus taken from man can excite tuberculosis in bovines when introduced in any of the various methods usually employed for inoculation. It has been shown, moreover, that the human bacillus thus acclimatized on the system of the cow gains in virulence by successive transference from one inoculated host to another until it becomes, in course of time, almost as virulent as that which is peculiar to the bovine tribe. In the face of such evidence it would be rash to argue that the difference between the human and the bovine organism is other than one of degree. Smith[3] reaffirms his views, expressed 4 years ago, pointing out the differences in the biologic characteristics of the two forms of the bacillus, and stating that we must not assume indiscriminate transference of the bovine form of the disease to man, nor that the human body cannot be invaded. De Schweinitz, Dorset, and Schroeder[1] publish some facts that show that the tubercle bacillus of human origin may cause tuberculosis in cattle, and that the morphology and virulence of the tubercle bacilli from various sources are greatly influenced by their surroundings. Adami[2] believes that the human tubercle bacillus differs from the bovine bacillus in many ways, but not sufficiently to permit us to conclude that they are distinct species; at most they are different races of tubercle bacilli which by growth in the bodies of animals of different species take on different characteristics peculiar to the species. The few cases of wound infection reported show that it is possible for man to become infected with the bovine bacillus, which, however, shows but slight virulence for man. It is believed that through the agency of milk young children may acquire intestinal tuberculosis, but that the trustworthy cases reported are few, and in these the infecting cattle were extensively diseased. [The evidence at hand is distinctly adverse to the tenability of the position of Koch, the prevailing opinion being that expressed by Ravenel, Hamilton, and others.]

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[1] Proc. Path. Soc. of Phila., new series, vol. v, 181, 1902.

[2] Brit. Med. Jour., II, 944, 1902.

[3] Med. News, Feb. 22, 1902.

Tuberculosis of the Skin Following Accidental Inoculation with the Bovine Tubercle Bacillus.—Ravenel[3] reports an additional case occurring in the person of a physician. Infection occurred while he was performing necropsies on cows dead of experimental tuberculosis. A scar developed 4 weeks subsequently, became prominent and sensitive, was excised, and being examined presented the typical picture of tuberculosis with tubercle bacilli. Inoculations of an emulsion made from the scar-tissue into guinea-pigs resulted in the prompt development of tuberculosis.

Direct Infection with Bacillus Tuberculosis.—Boyd[4] has seen 3 instances in which the tubercle bacillus gained admission to the human body through slight injuries in the hand. In each case lesions resulted, which were diagnosed after excision as characteristically tubercular. Primary union resulted in two of the cases, but in the third the disease continued to spread along the lymphatics in spite of all treatment. Eight operations were performed before the local disease was eradicated.

The Inoculability of Herbivora by Human Tuberculosis.—Arloing[5] refers to his previous work showing that goats and donkeys are inoculable with human tubercle bacilli, and he gives the results of some additional experiments, which comprise the inoculation of cows, sheep, kids, rabbits, and guinea-pigs, with a bouillon emulsion of a potato culture of human tubercle bacilli. All of the animals became affected with tuberculosis, and many of them had already died at the time of the report. From the tissues of the dead animals typical bacilli were isolated, and these, when inoculated into guinea-pigs, produced tuberculosis.

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[1] Amer. Med., IV, 850, 1902.

[2] Phila. Med. Jour., Feb 22, 1902.

[3] Univ. of Penna. Med. Bull., Feb., 1902, p. 453.

[4] Med. Press and Circ., July 10, 1901.

[5] Lyon méd., Dec. 1, 1902.

A case of conjunctival tuberculosis following a dog-bite and developing 3 months after the bite, in a child aged 5 years, is reported by Stutzer.[1] The dog is said to have eaten the placenta from a tuberculous cow immediately before biting the child.

Bacillus Tuberculosis in the Stools.—Anglade[2] has found Bacillus tuberculosis in very large numbers in the stools of tuberculous patients, even in cases in which there was no enteritis or intestinal ulceration. He urges the disinfection of the dejecta of tuberculous patients.

Tuberculosis of the Cervical and Bronchial Lymphatic Glands.—Walsham,[3] with a view to determine whether tubercle bacilli or their involution forms (Schrön's capsules, Cornet's spores, and Keimungsproducte) are always found in scrofulous glands, the phases the bacillus passes through in the glands, and the histologic changes produced by the tubercle bacillus in the glands, examined the cervical and bronchial glands obtained at necropsy from 27 subjects dead of pulmonary tuberculosis. In all the cases tubercle bacilli or Schrön's capsules were found; whence it is concluded that there can be no reasonable doubt that the so-called scrofulous gland is in reality tuberculous, and that the tubercle bacillus alone is responsible for the scrofulous change—though not for the suppuration that frequently occurs, this being due to mixed infection. With regard to the changes that the bacilli undergo in the glands, the following were encountered: Bacilli very long and thin, generally isolated, usually having a very beaded appearance and resembling streptococci; bacilli in bundles, generally of the short variety; bacilli with buds generally situated at one end, though occasionally two or three are found in the length of the bacillus; bacilli bent at a right angle, the two limbs of which are unequal—a very common form; bacilli with small offshoots from the side, mostly nearer one end than the other—a rare form; bacilli that took the staining reagent very badly, showing only a slight pinkish tint—probably bacilli in a very early stage of disintegration; and Schrön's capsules—mostly small oval or round bodies requiring an oil-immersion lens ($\frac{1}{12}$ inch) for their detection, generally found only in the lymphatics at the periphery of the gland, the round form usually staining light pink (with carbol-fuchsin), the oval form a deeper red. With regard to the changes produced in the gland by the tubercle bacillus it is said that the first change is a hyperemia and a proliferation of the adenoid reticulum, with a destruction or conversion, or, at any rate, a disappearance of the lymphoid cells. In other cases the only change consists of hyperplasia—the transformation of the normal gland tissue into large-celled tissue. This so-called large-celled hyperplasia was definitely proved to be tuberculous by the detection therein of the tubercle bacillus. It is believed that many scrofulous cervical glands become infected by way of the tonsils, and without ignoring inhalation as the principal channel of

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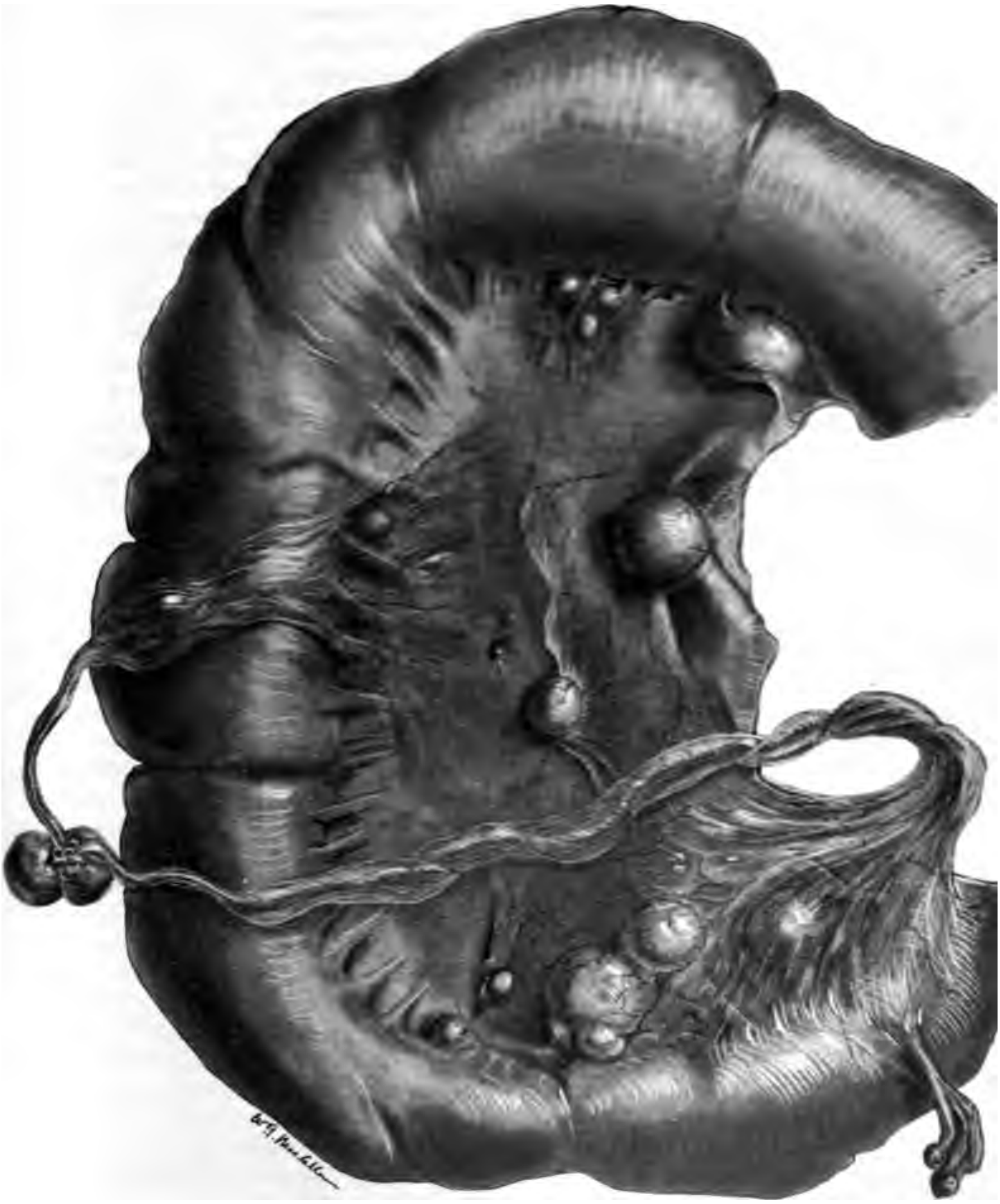
[1] Beiträge zur Augenheilk., xxx, 1901.

[2] Rev. de la tuberc., Aug., 1901.

[3] Jour. of Path. and Bact., vii, 409, 1901.

PLATE I.



Portion of the intestine, natural size, with its mesentery, from which arise the sessile and pedunculated nodules describ
(MacCallum, in Johns Hopkins Hosp. Bull., Sept., 1901).

tuberculous infection of the lungs, it is suggested that in some cases the lung may become infected by the gradual extension of the bacilli downward with the lymph-stream from infected cervical glands.

Pendulous Tubercles in the Peritoneum.—MacCallum[1] reports a case of the rare condition of pendulous tuberculosis occurring in the peritoneal cavity of a woman aged 38 years (see Plate 1). But one other case of this condition, corresponding with the perlsucht of lower animals, was found in the literature, the case reported by Bizzozero.[2]

Experimental Tuberculosis of the Suprarenal Capsule in Relation to Addison's Disease.—Inasmuch as the most frequent anatomic lesion in Addison's disease is tuberculosis of the suprarenal gland, De Vecchi[3] was led to inject tuberculous material into the capsules of rabbits. The animals lost 50 % in weight, there was slight fever and progressive diminution in the number of the red corpuscles; sometimes tremor and paresis of the extremities, but no discoloration of the skin and mucous membranes, nor dystrophy of the fur. Examination showed the capsules to be partially or completely caseated, the cortical substance less so than the medulla. The latter presented many epithelioid and giant-cells, the former considerable connective-tissue proliferation. The solar plexus, the splanchnic nerve, and the cervical and thoracic sympathetic ganglion were negative, except in one case, in which the celiac ganglion was tubercular. The cells of the gray matter of the central nervous system showed grave degenerative changes, which were attributed to a general intoxication with the tubercular toxin rather than to changes in the sympathetic system. The heart, liver, and kidneys also showed degenerative lesions. From the diminution of red blood-cells and the activity present in the bone-marrow it was suggested that the capsule possessed hemolytic properties, especially as a large amount of detritus accumulated in the spleen gave the iron reaction. The condition here produced seems to be due to two factors: the destruction of the suprarenal gland and the tubercular intoxication.

Tuberculosis and Amyloid Degeneration.—To determine the frequency of amyloid degeneration in cases of tuberculosis, Jahn[4] made careful statistical studies of 6320 autopsies conducted under his supervision: 2058 (32.5 %) showed tuberculosis, of which 165 were cases of latent tuberculosis, 1893 cases of acute tuberculosis. There were 1357 males and 701 females. The primary lesions occurred in the lungs in 1528 cases, in the bones and joints in 71, in the genitourinary tract in 42, in the adrenal bodies in 21, in the brain in 7, and in the intestines in 43. Intestinal lesions secondary to lung lesions were found in 966 cases. Amyloid degeneration was present in 105 cases; 79 of these were secondary to tuberculosis of the lungs, 19 to tuberculosis of the bones; 7 showed no tuberculosis.

Actinomycosis Hominis and the Differential Macroscopic Diagnosis between Actinomycotic and Tuberculous Peribronchitis.—Fütterer[5] reports a case of actinomycosis hominis, in which the diagnosis, based upon the finding of the ray fungi, was made during the life of the patient The clinical symptoms pointed to affection of some of the abdominal contents; the right lung also was thought to be affected. The necropsy revealed actinomycosis of the heart, lungs, diaphragm, and liver. There were two nodules found on the tricuspid valve. The left lung had but few areas, but the right lung, especially in its lower part, was changed to a cicatricial and pneumonic actinomycotic mass. Certain areas of the diaphragm were completely destroyed, being replaced by yellow and rust-colored masses. The liver also, especially in its upper and lowermost parts, was invaded by the yellow points. Abscesses were found in the lung and the liver, and at one point a perforation was seen in the diaphragm, a communication between the two organs being thus formed. The microscopic examination confirmed the macroscopic appearance. Fütterer differentiates between actinomycotic and tubercular peribronchitis, which, in his opinion, is not difficult, and may be done without resorting to the microscope.

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[1] Johns Hopkins Hosp. Bull., xv, 293, 1901.
[2] Morgagni, ix, 1867.
[3] Med. News, Nov. 2, 1901.
[4] Münch. med. Woch., Jan. 14, 1902.
[5] N. Y. Med. Jour., 1901, Aug. 24.

Eggs as a Medium for Cultivating Bacillus Tuberculosis.—To cultivate the bacillus of tuberculosis, Dorset[1] has used 3 different mediums, consisting of portions of hens' eggs, as follows: (1) The whole egg-contents, white and yolk mixed; (2) the yolk of the egg separated as far as possible from the white; (3) the white of the egg alone. When using the medium made from the white of egg, the results were always negative. Using the other mediums the growths resulted quickly and with comparative certainty, and as the medium is so easily prepared, he considers it of great value in experimental work in connection with the study of the tuberculosis bacillus.

The Etiologic Significance of the Acid-resisting Group of Bacteria and the Evidence in Favor of their Botanic Relation to Bacillus Tuberculosis.—Abbott and Gildersleeve,[2] from an exhaustive study of the subject, draw the following conclusions: (1) The majority of the acid-resisting bacteria may be distinguished from true tubercle bacilli by their inability to resist decolorization by a 30 % solution of nitric acid in water. (2) Some of the acid-resisting bacteria are capable of causing in rabbits and guinea-pigs nodular lesions suggestive of tubercles, which lesions, though often very like tubercles in their histologic structures, may nevertheless usually be distinguished from them by the following peculiarities: (*a*) when occurring as a result of intravenous inoculation they are always seen in the kidneys, only occasionally in the lungs, and practically not at all in the other organs; (*b*) they constitute a localized lesion, having no tendency to dissemination, metastasis, or progressive destruction of tissue by caseation; (*c*) they tend to terminate in suppuration or organization rather than in progressive caseation, as is the case with true tubercles; (*d*) they are more commonly and conspicuously marked by the actinomyces type of development of the organisms than is the case with true tubercles, and these actinomyces are less resistant to decolorization by strong acid solutions than are those occasionally seen in tubercles. (3) By subcutaneous, intravenous, and intrapulmonary inoculation of hogs (4) and calves (15) the typical members of the acid-resisting group are incapable of causing lesions in any way suggestive of those resulting from similar inoculations of the same animals with true tubercle bacilli. (4) Though occasionally present in dairy products, they are to be regarded as of no significance, etiologically speaking, but may be considered as accidental contaminations from the surroundings and not as evidence of disease of the animals. (5) The designation "bacillus" as applied to this group of bacteria and to the exciter of tuberculosis is a misnomer; they are more correctly classified as actinomyces.

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[1] Amer. Med., April 5, 1902, p. 555.
[2] Univ. of Penna. Med. Bull., xv, 106, 1902.

Acid-proof Bacilli in 5 Cases of Pulmonary Gangrene.—Ophüls[1] reports 5 cases of gangrene of the lung from which acid-proof bacilli were isolated. Reviewing the literature, he concludes that pulmonary gangrene is very probably always due to mixed infection with pyogenic cocci and other bacteria of a more saprophytic nature; that quite frequently the latter belong to the class of actinomyces (including in this class all branching bacteria like the tubercle bacillus, diphtheria bacillus, etc.), and that they may be more or less acid-proof. It is stated also that the gangrenous process is always accompanied by pneumonic processes, which in the more chronic cases usually appear in the form of catarrhal pneumonia with carnification.

Infectiousness of Dwellings Occupied by Consumptive Persons.—Coates[2] investigated the dust of houses occupied by tuberculous patients, concerning its infectiousness. The investigation comprehended: (1) A study of dust from houses in a dirty condition, and in which the consumptive patients exercised no precaution to prevent infection of the atmosphere by the expectoration, but spat freely on the floor, or in a pocket handkerchief, etc.; (2) dusts from houses in a very clean condition, but in which the consumptive patient did not exercise great care concerning the disposal of sputa; (3) dusts from houses in which there have been no cases of tuberculosis for several years. Dust was collected from situations in which dust from the atmosphere had settled naturally, and where there would be a likelihood of direct contamination with expectoration or by infected articles, in part by means of a sterilized spatula, and in part by a sterilized sponge. Tubercle bacilli were demonstrated microscopically in only 2 cases out of a very large number of film preparations, and this method of study was therefore abandoned for the method of guinea-pig inoculation. Twenty-three houses of class 1 were examined, definite results being arrived at in 21 cases. Of 52 guinea-pigs inoculated, 20 died in a few days after inoculation, 32 were killed 4 or 5 weeks after inoculation; 17 were found to be tuberculous and 15 healthy. The greatest infectivity of the dust was that nearest the floor, but taken from parts of the room where direct infection could not have taken place. Of class 2, ten houses were examined, 20 guinea-pigs being inoculated. Three died of septicemia, 6 developed tuberculosis, and 11 were found healthy at the postmortem examination. Of the 10 rooms, dust capable of producing tuberculosis was found in 5. Thus even in the cleanest of houses infectious dust can be found in 50 % of cases when the phthisical occupant is using his pocket handkerchief or floor for a receptacle for sputum. Of class 3, ten houses were examined, and in no case was tuberculous dust discovered. Twenty guinea-pigs were inoculated, 6 dying from sepsis. The remaining 14 were killed 4 or 5 weeks after infection, but all were free from tuberculosis. These results confirm those of Cornet made 11 years ago, and show that, while infection by the tubercle bacillus may result from ingestion, in such a vitiated atmosphere the danger of inhaling tubercle bacilli is great. He advises the thorough disinfection of premises previously occupied by tuberculous patients, and believes that should health boards adopt energetic measures for the destruction of the infectious elements in such dwellings, much avoidable illness and considerable loss of life might be prevented.

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[1] Jour. of Med. Research, VIII, 342, 1902.

[2] Therap. Monthly, Jan., 1902.

TYPHOID FEVER.

The Significance of the Typhoid Serum Reaction in the Offspring of Patients Suffering with Typhoid Fever.—Bolton[1] reviews the literature and reports 3 personal observations. In 2 there was neither infection of the fetus nor a serum reaction with the blood of the fetus; in the third there were both infection of the fetus with Bacillus typhosus and a serum reaction. It is believed that inasmuch as in the single case in which there was a serum reaction the fetus had become infected with Bacillus typhosus, the bacillus probably had formed its own agglutinin, and that, although experimental evidence shows that on repeated inoculation of the mother a little agglutinin may pass to the fetus, there is no case on record in which the child gave a positive reaction in which the organs have been proved free from Bacillus typhosus. Until this is shown, the passage of agglutinin through the placenta in the human subject must be considered unproved. On the other hand, one should remember that agglutinin has been proved to exist in the milk and colostrum of a woman whose blood gives the serum reaction, and it has also been proved that it is absorbed from the milk by the suckling child.

Pseudo-clumping in Cultures of the Typhoid Bacillus.—Savage[2] details the results of a series of experiments undertaken to determine the cause of the spontaneous clumping of typhoid bacilli sometimes observed in bouillon cultures. Having eliminated calcium carbonate and particles of foreign matter and debris as possible factors in the production of the phenomenon, he finally reached the conclusion that susceptibility of the race of Bacillus typhosus is the most important factor—a susceptibility which can be artificially induced or naturally acquired (by long cultivation outside the body), and which when naturally acquired is with difficulty got rid of. A second factor is the constitution of the broth in which the bacillus is grown—due mainly to the Liebig's extract or meat used in preparing the broth. The important result of the investigations is the conclusion that there is in bouillon a substance or substances capable of causing agglutination in susceptible races of bacilli. There is no evidence to show that the bacilli secrete a specific agglutinating secretion, or that the specific agglutinins are a product of the bacillus and not of the cells of the body. Apparently clumping is not only caused by the specific agglutinins, but it can be set up in a feeble degree by a number of chemic substances.

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[1] Jour. of Path. and Bact., VII, 137, 1901.

[2] Jour. of Path. and Bact., VII, 388, 1901.

Brain Abscess in Typhoid Fever Due to Bacillus Typhosus.—McClintock[1] reports a case of abscess of the brain from which Bacillus typhosus in pure culture was isolated (believed to be the only case recorded). A review of the literature disclosed reports of 19 cases of meningitis in which Bacillus typhosus was present, in the great majority in pure culture, and 5 cases of brain abscess in none of which the bacillus was found.

A new and simple medium for differentiating the colonies of typhoid, colon, and allied bacilli is described by Hiss.[2] Of several formulas given, the simplest is, agar 15 grams, Liebig's extract of beef 5 grams, and distilled water 1000 cc. Other formulas consisting in the addition to the simpler formula of sodium chlorid, dextrose, and gelatin, alone or in combination, are given, but the simplest usually suffices. Grown in this medium the deep typhoid colonies are of irregular shape and have well-marked threads of bacilli given off from them. They vary in size according to the composition of the medium and the number of colonies in the plate. Their texture is generally loose, and their color often light green in the small colonies, becoming darker in the larger colonies. Surface colonies may be quite flat and homogeneous, or they may show the denser center with threads spreading out from it. The deep colonies are often not more than one-fifth, and sometimes even less than one-tenth, as large as the deep colon colonies. The typical deep colon colonies are denser and darker as well as larger than the typhoid colonies, they do not have irregular outgrowths and fringing threads, and they are readily distinguished from the typhoid colonies. The surface colon colonies in some of the mediums are quite characteristic, being large disc-shaped, of a deep color nearly to the periphery, terminating in a well-marked light thin zone. This is especially the case in the simpler agar mediums.

DIPHTHERIA.

Persistence of Varieties of the Diphtheria Bacillus and of Diphtheria-like Bacilli.—Anna W. Williams[3] gives the results of (1) a study of diphtheria and diphtheria-like bacilli found in a series of clinically typical diphtherias; (2) a study of the diphtheria and diphtheria-like bacilli found in healthy and diseased throats in a town during an epidemic of diphtheria; (3) a study of diphtheria and diphtheria-like bacilli found in sore throats during an epidemic of diphtheria at a home for destitute children; (4) a study of pure cultures of diphtheria and diphtheria-like bacilli from sources other than those mentioned. In a series of 10 cases of clinically typical diphtheria only one variety of the specifically virulent diphtheria bacillus was obtained from the throat of each patient throughout the course of the disease and until suspicious bacilli disappeared. From different parts of the same patient only one variety of specifically virulent diphtheria bacillus was obtained. Pseudo-varieties were found no more frequently at the end than at the beginning of the disease. Pure cultures of the bacilli continued to show the same characteristics for many cultural generations. From the foregoing it is inferred that specifically virulent diphtheria bacilli do not change readily, if ever, into any form of nonvirulent diphtheria bacilli in throats or noses of persons during an attack of diphtheria. The second series of investigations lead to the following conclusions: (1) In normal throats different varieties of diphtheria-like bacilli may be found in the same throat; (2) many throats in a single institution may contain many diptheria-like bacilli with no case of diphtheria resulting; (3) there are many distinct varieties of diphtheria-like bacilli, all of which in serial pure cultures retain the characteristics of the original culture—from which it is inferred that nonvirulent nontypical forms of diphtheria bacilli do not change readily, if at all, into virulent forms in healthy throats. The third series of investigations lead to the following conclusions: (1) In cases of diphtheria apparently contracted from the same source the same variety of virulent diphtheria bacillus was found in each case; (2) this variety remained the only variety of virulent diphtheria bacillus found until its disappearance; (3) no gradations in pseudo-types were found, the same type found in the first examination, continuing to be present in later ones; and (4) pure cultures continued to show characteristics similar to the original cultures—from which it is inferred that not only does a variety of diphtheria bacillus retain its characteristics for some time in the same throat, but that it may be transferred to other throats without losing its individuality. The different varieties of diphtheria and diphtheria-like bacilli are described in detail, and it is said that since there are so many different forms or varieties of diphtheria-like bacilli, it is quite possible that some of them are so nearly related to the diphtheria bacillus that under certain conditions they readily develop its characteristics—otherwise it seems impossible to explain the apparent discrepancies in the results obtained by different observers. It is believed that inasmuch as many nonvirulent diphtheria-like organisms retain their characteristics under various artificial and natural conditions, they may be regarded from a public health standpoint as harmless; and it is further believed that the morphologically typical diphtheria bacillus is a species distinct from the atypical diphtheria-like bacilli and so-called pseudo-forms, and that it has many true morphologic varieties or subspecies which, while showing transitory ontogenic variations due to changes in environment and life habit, have more or less persistent phylogenic characteristics that reappear when the organism is placed in a previous environment.

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[1] Am. Jour. Med. Sci., CXXIII, 595, 1902.
[2] Jour. of Med. Research, VIII, 148, 1902.
[3] Jour. of Med. Research, VIII, 83, 1902.

The Pathology of Postdiphtheric Paralysis.—Laslett[1] believes that in the paralytic stage of diphtheria the only important change is in the peripheral nerves; that if there are any changes in the cells during the paralytic stage, they do not stand in causal relation to the parenchymatous degeneration in the nerves; and that, therefore, the parenchymatous degeneration of the nerve-fibers must be regarded as the primary lesion.

Branching in Bacteria with Special Reference to Bacillus Diphtheriæ.—Hill[2] reviews thoroughly the literature concerning branching in bacteria and gives the results of his own studies, which are thought to justify the following statements: (1) Passive degeneration changes in dead or dying bacilli may give rise in this species to slight irregular projections which distantly simulate branches, using this term in a wide sense. (2) As a part of the active development of the diphtheria bacillus, active branching by apparent budding, ending in the production of an oval elliptic body, probably capable itself of further development and the production of new rods, may occur in very young cultures, the parent stem then degenerating. (3) As a part of the active development of the diphtheria bacillus, branching similar to that described, but terminating in an ordinary diphtheria rod-like body, and without any degeneration of the parent stem at the point of origin, may occur within 24 hours of inoculation; and this new rod may segment in the ordinary way, or itself produce branches, terminating in rods similar to itself or in oval bodies such as are described above. (4) Various modifications of all the processes of branching described probably exist. (5) The origin of the active multiplicative branching may be revisionary or evolutionary or merely due to special conditions of growth not understood at present. According to the latter view, the active forces usually resulting in fission may at times undergo a lessening of tension or some other modification which results in a change in direction of their activities. Whether such a change is regressive, "a degeneration of forces," as phrased by Sedgwick, or progressive, an "ascension of forces," has not yet been determined.

An interesting **epidemic of milk diphtheria** is reported by Dean and Todd.[3] Investigation showed that the patients had obtained their milk-supply from two cows which were found to be suffering from an eruptive disease of the udder; and both from the milk and from the lesions cultures of virulent diphtheria bacilli were isolated. At first, it was thought that these udder lesions were specific diphtheric infection, but further investigation weakened this view. It was found: (1) That in a cow intentionally submitted to infection from the diseased cows, and in which there occurred the eruptive condition of the udder, neither in the vesicular nor in the ulcerative stage of the disease could diphtheria bacilli be demonstrated; (2) that in calves infected with the eruptive disease no diphtheria bacilli could be demonstrated; (3) that in a calf, in spite of the fact that it had received 10,000 units of diphtheria antitoxin, the vesicular eruption was experimentally produced. The last is probably the strongest point in support of the dual nature of the condition in the cows. It is conceivable that pathologic lesions in the cow such as those described, if infected with the diphtheria bacillus, might form a suitable nidus for its growth, and might permit of the infection of large quantities of milk over a considerable period. The disgusting habit of spitting on the hands before milking, which the milkers in certain parts of England have, would easily account for the infection of the lesions, even in the absence of obvious diphtheria in the cow-man. It is possible that a profound change in the virulence of the diphtheria bacillus for the human subject may be effected by passage through the cow. The experiments showed that the eruptive disease in the udder was not genuine cowpox.

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[1] Jour. of Path. and Bact., VIII, 228, 1902.

[2] Jour. of Med. Research, VII, 115, 1902.

[3] Jour. of Hyg., 1902, No. 2, vol. II

SCARLET FEVER.

The Bacteriology of Scarlet Fever.—Baginsky and Monti[1] have made a careful bacteriologic study of scarlet fever, examining the pharynx, occasionally the fluid obtained by lumbar puncture, and the urine during life, as well as the blood and the organs after death. In 411 cases smears from the pharynx showed the presence of streptococci in short and long chains, almost constantly associated with other cocci, particularly the pneumococcus, staphylococcus, and other diplococci, and at times with yeasts, leptothrix, bacilli, spirochetæ, and sarcinæ. In 22 the diphtheria bacillus was also present. In 138 cases culturally examined the streptococcus was also found—in all but 4 in mixed cultures. The lumbar fluid in the 2 cases examined contained streptococci, as did also the urine. From the autopsy material studied the streptococcus was isolated in every instance, and nearly always in pure culture. The virulence of the organism was most variable, but by passage it was possible greatly to intensify it. Agglutination with the blood-serum of children just convalescing from scarlet fever was not obtained. Attempts were made to protect animals against the streptococcus by injecting emulsions of organs, such as the spleen, liver, brain, kidney, and bone-marrow, without any result. The injection of sterile blood-serum from convalescent patients seemed in 2 instances to prolong life; in 4 it was entirely ineffectual. The article contains no conclusions regarding the relationship of the streptococcus to the disease. In the long appendix of 42 pages the histories of 82 cases are given.

The Leukocytosis of Scarlet Fever and its Complications.—Bowie,[2] from an exhaustive study of the subject, states that practically all cases of scarlet fever show leukocytosis, which begins in the incubation period, very shortly after infection, reaches its maximum at or shortly after the height of severity of the disease, and then gradually sinks to normal. In simple uncomplicated cases the maximum is reached during the first week, and the normal generally some time during the first 3 weeks. The more severe the case, the higher is the leukocytosis and the longer it lasts; the slighter the case, the less the leukocytosis and the shorter time it lasts. A favorable case in any one variety of the disease (*e. g.*, simple, anginose, etc.) has a higher leukocytosis than an unfavorable case of the same variety. The fever—that is, the temperature—has no direct effect on the leukocytosis. The polymorphonuclear leukocytes are increased relatively and absolutely at first and then fall to the normal, the lymphocytes acting inversely to this; this cycle of events occurs in simple cases within 3 weeks. Eosinophiles are diminished at the onset of the fever. They increase rapidly in simple favorable cases till the height of the disease is past, then diminish, and finally reach the normal some time after the sum total leukocytosis has disappeared—in short, when the poison has been eliminated. The more severe the case, the longer are the eosinophiles subnormal before they rise again; in fatal cases they never rise, but sink rapidly toward zero and remain there. The leukocytes in complications go through a cycle of events similar in all respects to that of the primary fever, as regards both sum total and differential leukocytosis, and the same laws govern the behavior of the leukocytes in both cases. With regard to diagnosis, it is said that a simple counting of the leukocytes is of little value, since most of the febrile conditions with which scarlet fever is likely to be confounded have a leukocytosis of their own. Measles, which is classically represented as the disease from which scarlatina is to be differentiated by the leukocyte count, is practically never confounded with this disease. The differential count of the leukocytes, however, is of value, since scarlatina is one of the few infectious diseases in which one finds an increase in the eosinophiles early in the disease, and a persistent increase for some time. Thus, tonsillitis and septic conditions may be readily differentiated from mild cases of scarlatina by the long-continued eosinophilia.

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[1] Arch. f. Kinderheilk., 1902, Bd. xxxiii, Hefte 1 u. 2.
[2] Jour. of Path. and Bact., viii, 82, 1902.

YELLOW FEVER.

The Etiology, Propagation, and Prevention of Yellow Fever.—Walter Reed[1] published a summary of the work of the United States Army Commission, which was organized in May, 1900, for the purpose of studying yellow fever in Cuba. Their first work consisted in endeavoring to isolate Bacillus icteroides of Sanarelli; but in 24 carefully studied cases the results were negative. Moreover, a careful study of 11 autopsies was equally barren as to the presence of any particular microorganism. This was also true of fresh and stained specimens of blood. Although a microscopic and bacteriologic study of the blood was negative, the infective agent must, nevertheless, be present in the blood; for the Commission were able to produce the disease by the injection of small quantities of blood into nonimmune individuals. They found that the infective agent was present at least during the first, second, and third day of the attack. They were even able to produce the disease by the injection of a small quantity of bacteria-free serum-filtrate obtained by passing the diluted serum through a Berkefeld filter. As to the propagation of yellow fever, the Commission proved conclusively that it is not conveyed by vomitus. Nonimmune individuals slept every night for 21 days in the very garments worn by yellow fever patients throughout the entire attack, besides making use of their much-soiled pillow-slips, sheets, and blankets, without acquiring the disease. The series of nonimmune individuals was then allowed to be bitten by mosquitos which had previously fed upon the blood of patients ill with yellow fever in the city of Havana. The species of mosquito was Stegomyia fasciata. Positive results were obtained in 14 cases. Guiteras[1] has since published corroborative results. Of 17 patients bitten by infected Stegomyia, 8 developed the disease, which in 3 ended fatally. It seems that an interval of about 12 days after contamination is necessary before the infected insect is capable of conveying the disease to a susceptible individual. Furthermore, mosquitos kept for periods varying from 39 to 57 days after contamination are still capable of conveying the disease, and infected Stegomyia may survive for a period of at least 71 days. These most important studies lead to the natural conclusion enunciated by the Commission, that the "spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitos, and the protection of the sick from the bites of these insects." The article contains tables showing the good results obtained in Havana. A supplemental note on the subject with references to the literature appears in "American Medicine," vol. III, 301, 1902.

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[1] Jour. of Hyg., 1902, No. 2, vol. II.

MALARIA.

Inoculations Against Malaria.—Kuhn[2] states that since 1899 he has maintained that malaria and horse sickness are but narrowly separated from each other, and that for purposes of his experiments he considered them identical diseases. He points out that neither disease is immediately infectious, that both appear with the rains and cease with the onset of cold weather, that mosquitos probably play a like part in both, and that in some respects the two diseases resemble each other clinically. To the objection that horse sickness is not prevalent in certain countries severely malarious, he replies by suggesting that minute investigations would probably disclose the fact that horse sickness either prevails everywhere in tropical malarious countries, or has yielded to the gradual "salting process." Preparing, then, a serum curative of horse sickness, he employed it in treating malaria, with happy results. One cubic centimeter or more of the serum was injected into forearms of malarious subjects—both white men and the natives of German southwest Africa. At the site of the inoculations a slight and somewhat painful swelling sometimes arose, but it disappeared entirely in several days; in some cases urticaria developed. Although the patients suffered with unmistakable malaria and took no quinin, all forms of the disease yielded to the injections. In all cases the cold sensations of the first stage were diminished in intensity. The effects depended somewhat upon whether or not the patients had had malaria. In the case of the natives and old residents the inoculations in many instances stopped the attacks at once; in newly arrived immigrants, who previously had not had malaria, and in children, after the inoculations there occurred on an average 4 or 5 attacks, the last always being the weakest. Between these extremes were the cases of the white settlers, in whom usually 1 to 3 attacks followed the inoculations. Relapses were found to be less frequent in the cases of quotidian or tropical malaria than in the cases of tertian or quartan fever, and the inoculations were found to be more efficacious when given during an attack than when given during the intermissions. Kuhn claims that his inoculation treatment serves as a substitute for quinin; that inasmuch as quinin is believed to induce blackwater fever in some cases, this will be avoided by the use of his serum; that the inoculations tend to prevent relapses; and that they prevent fresh infections. [Not at present to point out some of the breaks in Kuhn's chain of evidence, it suffices to mention the suggestiveness of his reasoning and experiments, and to hope for confirmation of his results. In particular, it would appear to be incumbent upon those enjoying the opportunity to ascertain the nature of horse sickness.]

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[1] Amer. Med., Nov. 23, 1901.

[2] Arch. f. Schiffs- und Tropen-Hygiene, Nov., 1901; Brit. Med. Jour., Jan., 1902.

Malarial Nephritis.—Ewing[1] gives a review of the literature and the results of his study of 14 fatal cases of malaria, from which he concludes that 3 main types of acute renal lesions occur in malaria: (1) Acute degeneration of toxic origin, often reaching a degree in which exudation of blood-serum into the tubules is added. This lesion is responsible for the vast majority of cases of albuminuria in malaria. (2) An extreme form of acute degeneration, with focal necroses, numerous hemorrhages, and exudation into the tubules of blood-serum and blood-pigment. This lesion is seen in cases of hemoglobinuric malarial fever, and it has not yet been found associated with an excessive number of parasites in the capillary vessels. (3) Massing of parasites in the renal capillaries, with extreme degeneration of parenchyma cells, multiple hemorrhages, and exudation of blood-serum into the tubules. It seems that this type of lesion can occur only in severe estivoautumnal infections. There is anatomic evidence that in the pernicious estivoautumnal cases the 3 types of lesions may be variously combined, but no good reason for believing that with the benign tertian infection occurring in this latitude any other than the first-mentioned type can exist. Details are given of a case of malaria, developing in the vicinity of New York, presenting the symptoms of acute hemorrhagic nephritis with partial suppression of urine, and giving clear microscopic evidence of extreme massing of parasites in the kidney with various mechanical lesions resulting from their presence—whence the conclusion that malarial nephritis may be caused principally or in large part by the presence of an excessive number of parasites in the renal capillaries.

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[1] Am. Jour. Med. Sci., CXII, 426, 1901.

Differential Staining of Blood-films and Malarial Parasites.—Wright[1] advocates the use of the following blood-staining method, which he states gives not only a differential staining for the chromatin and the cytoplasm of malarial parasites, but is also a general staining method for blood and, as such, is superior to Jenner's method, in that it sharply differentiates the nuclei and the cytoplasm of the lymphocytes and mononuclear leukocytes, as well as the granulations of the leukocytes in general. The stain is prepared by making a 0.5 % solution of sodium bicarbonate in water in an Erlenmeyer flask and adding to this 1 % of Grubler's methylene-blue. The mixture is then steamed in an Arnold steam sterilizer for 1 hour, counting the time after "steam is up." When cold, without filtering, the mixture is poured into a large dish, and to it is added, stirring meanwhile, a sufficient quantity (usually about 500 cc.) of a 1 : 1000 solution of eosin (Grubler's yellowish, soluble in water) until the mixture, losing its bluish color, becomes purple in color, and a scum with yellowish metallic luster forms on the surface, while on close inspection a finely granular black precipitate appears in suspension. The precipitate is collected on a filter and without washing is allowed to dry thereon. When thoroughly dry, a saturated solution in pure methylic alcohol is made (0.3 gm. of the dry precipitate will thoroughly saturate 100 cc. of the methylic alcohol). The saturated alcoholic solution is next filtered, and to the filtrate is added 25 % of methylic alcohol. This forms the staining solution. The technic of staining the blood-films is as follows: The film, having been dried in the air, is covered with the stain for 1 minute, and then to the staining fluid on the cover-glass is added, drop by drop, sufficient water (usually about 8 to 10 drops) until it becomes semi-translucent and a reddish tint becomes visible at the margins, while a metallic luster forms on the surface. This is allowed to remain for 2 or 3 minutes. The cover-glass is then washed in water until the film has a yellowish or pinkish tint in the thinner and better spread portions. Then it is dried in filter-paper and mounted in balsam.

VARIOLA AND VACCINIA.

Variola.—Ewing[2] gives a preliminary report of his study of 29 cases of smallpox, which study comprised the pathologic anatomy and histology of the lesions of the skin and internal organs, the routine examination of the heart's blood and the skin lesions by ordinary methods, and by special bacteriologic methods, and experimental variola. The skin lesions revealed the presence of vaccine bodies in all stages of the specific lesions—their numbers being proportionate to the hemorrhagic character of the lesion. In addition, streptococci were present in all the specific lesions at a very early stage, frequently in enormous numbers, but very often scantily; but sometimes they could not be detected in the smears of vesicles and even of pustules. The next most common bacterial form was a fine bacillus resembling in general a minute Klebs-Löffler bacillus. Examination of the blood of 12 cases confirmed the previous work of Courmont and Montagnard,[1] revealing the presence of from 30 % to 50 % of large and medium-sized mononuclear cells and some myelocytes, which with distinct leukocytosis and active fever is believed to be rather pathognomonic of the disease. Ewing states that, at the present time, while it hardly seems possible that the streptococcus is the specific contagium of variola, one cannot fail to be strongly impressed by the fact that this microorganism is present at all stages of the disease and in numbers proportional to its severity. It is difficult to reconcile the principles governing secondary infection by streptococcus with the presence of this bacterium in the blood during life, and with its enormous numbers in the blood very shortly after death in the primary hemorrhagic cases. In these cases it cannot be doubted that the streptococcus completely overshadows any other bacterial agent that may be present, and the idea of a secondary infection in such cases does not seem reasonable. Ewing believes, therefore, that the condition in these cases should be regarded rather as a symbiosis of the streptococcus and the specific agent, from a very early period, so early as to be indistinguishable in these cases from a primary invasion. While not inclined to urge that the streptococcus is the specific agent in variola, he believes that it will always remain impossible to reproduce the disease, especially the fulminant types, without the aid of the streptococcus

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[1] Jour. of Med. Research, VII, 138, 1902.

[2] Trans. Assoc. Am. Phys., XVII, 208, 1902.

Differentiating between Variola and Varicella by Inoculating Monkeys.—Park,[2] as a result of an experimental study of the subject, concludes that virus obtained from scraping developed vesicles or pustules of smallpox when inoculated into the skin of the monkey in the manner in which human vaccination is performed, causes within 48 hours a local infection similar to a moderately severe vaccination with cowpox. Virus from well-developed vesicles and pustules in cases of chickenpox, however, does not infect monkeys. It is believed, therefore, that inoculation of monkeys affords a means, in doubtful cases, of differentiating between smallpox and chickenpox within a period of 3 days. While a single positive inoculation is sufficient to establish a diagnosis of smallpox, a negative result in one animal should be confirmed by a second inoculation in another animal before establishing a diagnosis of chickenpox, since it is possible that just as vaccination fails in men variolation may fail in monkeys.

The Pus and Blood in Variola.—Roger[3] gives the results of his study of the pus and blood in variola. In the pustule he found a larger number of mononuclear cells than in pyemia, acne vulgaris, and multiform erythema (in which condition staphylococci with polynuclear cells are found). In variola, especially in the early stage of the pustule, no microorganisms are present in the pus, which, however, contains small, roundish, and ovoid bodies that Roger believes are the specific agents of the disease. Examination of the blood-stained fluids given off in cases of hemorrhagic variola shows these small bodies or corpuscles, which are also present in the blood and in the early stage of the pustular eruption. As Courmont and Montagnard have pointed out, the leukocytosis of variola is of the mononuclear type—rather a myelocytosis which may be compared to that observed in lymphadenia. The polynuclear cells are always less numerous than in normal blood. They vary from 40 % to 50 %, and in severe cases, especially in hemorrhagic cases, they may fall as low as 20 % or even 14 %. Of the mononuclear cells the medium-sized are the most numerous—30 % to 40 %. Besides these there are large mononuclear neutrophiles (2 % to 10 %), a few eosinophiles (0.5 % to 1 %), and a mononuclear kind of cell whose nongranular protoplasm has an affinity for nuclear stains (2 % to 10 %). There are also transition forms and eosinophiles which later may be as numerous as 1.5 % to 3 % at first, followed by a diminution in the course of the disease, to again increase during convalescence. During the period of suppuration and when the pustules are drying a few white cells are found, either mononuclear or polynuclear, with basophile granules, but they are always few in number. The abnormal cells, abundant at first, persist during the stage of pustulation, but gradually diminish during convalescence. Still some may be found at the end of 40 days. The blood-formula of variola is believed to be so characteristic as to be of considerable assistance in the diagnosis, in differentiating it from measles and scarlatina. Varicella, however, shows changes analogous to those found in variola. But Roger considers this not remarkable, since varicella is due to organisms analogous to those of variola. The two diseases, however, differ so markedly clinically that there is little difficulty in arriving at a correct diagnosis.

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[1] Jour. de Phys. et de Path. gen., 557, 781, 1900.

[2] Trans. Assoc. Am. Phys., XVII, 217, 1902.

[3] Presse méd., No. 12, Feb. 8, 1902; Brit. Med. Jour., Mar. 15, 1902.

Tetanus and Vaccination.—McFarland,[1] from an analytic study of 95 cases of this rare complication, draws the following conclusions: (1) Tetanus is not a frequent complication of vaccination, a total of 95 cases having been collected; (2) the number of cases recently observed is out of all proportion to what has been observed heretofore; (3) the cases are chiefly American and occur scattered throughout the eastern United States and Canada; (4) they have nothing to do with atmospheric, telluric, or seasonal conditions; (5) they occur in small numbers after the use of various viruses; (6) an overwhelming proportion has occurred after the use of a particular virus; (7) the tetanus organism may be present in the virus in small numbers, being derived from the manure and hay; (8) occasionally the number of bacilli becomes greater than usual through carelessness or accident; and (9) the future avoidance of the complication must be sought for in greater care in the preparation of the vaccine virus. R. N. Willson,[2] having studied the same question, reaches the conclusion that in most, if not all, the cases of tetanus following vaccination, infection took place at the site of the vaccination. He believes that absolute and scientific proof of the exact time of infection, and the exact means, is impossible, and that the grouping of a large number of cases in a certain locality, and following the use of a certain make of vaccine virus, would tend at first sight to speak for a primary infection carried in with or by means of the vaccine virus into the system. On the other hand, Willson believes that a secondary infection, and one occurring as a rule about the time of the acme of the vaccinia, is indicated by the otherwise discordant chronic incubation period and acute symptoms, by the almost uniformly fatal termination, by the severity of the course of the disease, by the millions of normal vaccinations with the same virus, by the simultaneous deaths from tetanus known to be due to other causes, by the diminution in the number of such deaths now that continued aseptic care is more generally exercised, by the fact that in every case in which particulars are known overabundant opportunity was offered for such secondary infection, and, finally and most important, by the absolute failure of all bacteriologic and inoculation experiments on the lower animals and man to indicate the presence of the tetanus microorganisms or their toxin in the virus.

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[1] Jour. of Med. Research, VII, 474, 1902; Lancet, II, 1902; Medicine, June, 1902.
[2] Jour. Am. Med. Assoc., XXXVIII, 1147, 1222, 1902.

Dry Vaccine Points Versus Glycerinated Virus, from a Bacteriologic Standpoint.—Rosenau[1] and others have studied dry points versus glycerinated virus, from a bacteriologic standpoint. Of 41 dry points examined they found an average of 4807 bacteria per point; of 51 glycerinated tubes and capsules, an average of 2865 bacteria per vaccine. This is in excess of what a good glycerinated virus should contain. This difference does not justify the confidence placed in the glycerinated virus over the dry points as found upon the market, judging from the limited number of counts made. They found pus cocci in both the dry points and the glycerinated virus. In their opinion their tests demonstrate that some of the glycerinated virus is not kept a sufficient length of time before it is sold, and they conclude that glycerinated virus ought not to be discredited (its superiority has been amply demonstrated), but the practice of manufacturers who place an unripe product on the market should be condemned. Much of the vaccine sold must have had a high initial contamination to contain an average of 2865 bacteria per tube, and it is evident that too great a reliance was placed upon the glycerin.

PLAGUE.

The Pathology of Bubonic Plague.—Flexner[2] discusses the pathology of bubonic plague with particular reference to changes in the lymph-glands and the spleen, basing his statements upon the postmortem examination of 6 patients, and the records of many other cases. Characteristic changes are found in the lymphatic glands, which may be affected primarily or secondarily. Primary involvement may be of two kinds: of the first order, being the point of the original reception of the virus; of the second order, representing the extension to a second, contiguous group of glands. The buboes of the latter are less marked than of the former, and may be found in any or all glands within the body, and at a distance from the primary lesions, infection having followed through the blood circulation. They show slight swelling and congestion; their subcapsular sinuses are dilated, and are filled with numerous large cells, which are phagocytic to red corpuscles. The lymphatic cells throughout the glands tend to increase in number, and fine strands of fibrin form in places. The more intense the inflammation, the greater the changes named, going even as far as slight fragmentation. But few bacilli are to be found in these glands. In primary buboes the pathologic changes are much more intense, and also different in kind. Hemorrhages occur throughout the glands, and all the tissues, especially those of the medulla, show necrotic changes. The blood-vessels are filled with cellular and fibrinous thrombi. The periglandular tissue shows the same changes to some extent. The nerves are also involved. Bacilli are abundant within the swollen glands and in the periglandular tissues, occupying every available space. The buccal mucous membrane forms one of the portals of entry into the body of the plague bacilli. Of all the buccal structures, the tonsil seems to be most frequently the one primarily attacked; its changes are not unlike those described in the primary lymphatic bubo of the first order. There is congestion, hemorrhage, necrosis of cells, and extensive growth of bacilli. Many of the tonsillar cells are phagocytic for bacilli. Of this there can be no doubt, for the protoplasm of many of them are packed with characteristic organisms. The spleen is enlarged moderately, its consistence is somewhat diminished, and its color deeper than normal, but it is not the type of the acute splenic tumor of true septicemia. Flexner describes the minute changes that are found in the spleen, and then goes on to a consideration of the lesions of the disease produced experimentally in the guinea-pig. The disease in them shows either the picture of a bacteremia without macroscopic localization in the organs, and focal nodular localizations in the internal organs. In all cases marked local lesions at the site of inoculation and in the adjoining tissues occurred. He describes the macroscopic and microscopic changes, which do not differ greatly from the lesion found in the human organism. In man two main types of the plague, bubonic and pneumonic, may be considered as established.

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[1] Amer. Med., Ap. 19, 1902, p. 637.

[2] Univ. of Penna. Med. Bull., Aug., 1901, p. 205.

GONOCOCCUS.

The Gonococcus and its Toxin.—Eastman[1] considers the morphologic and biologic characteristics of the gonococcus as well as the methods of staining it. The position of the gonococcus, whether within or without the cell, depends entirely on the stage of the inflammation, but that it is found both within and without has been observed by many. In the acute stage the intracellular form predominates. As the disease subsides, many gonococci free from cells are found, the cells most likely having become disorganized, leaving the cocci free; they also begin to disappear, and as the chronic stage is reached but few gonococci are seen, and they are located in the epithelial cells. In undoubted gonorrheal pus the best routine stain is Löffler's methylene-blue, but in doubtful cases Gram's method should be applied to distinguish the organism from other diplococci. In some cases the cultural method will have to be used to make the diagnosis. The best solid medium to use is Löffler's or Koch's solidified serum smeared with fresh, sterile, human blood. The best fluid medium is ascitic fluid and peptonized bouillon, 1 part to 3, to which is added glucose to the proportion of 1 to 1000. He discusses the conditions in which the gonococcus is found, and conditions believed to be due to its toxin.

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[1] N. Y. Med. Jour., Sept. 28, 1901.

Biology of the Gonococcus.—It has been generally admitted that the best temperature for the development of the gonococcus is between 36° and 38° C., and that at 39° C. the organism loses its vitality. Nobl[1] publishes 5 cases of gonorrhea complicated by febrile affections (typhoid fever and pneumonia) in which, in spite of the prolonged temperature of 40° C., the gonococcus existing in the urethra of the patients lost none of its virulence nor resistance.

SCURVY.

The Etiology and Pathology of Scurvy.—Lamb[2] refers to the 3 hypotheses recently advanced to account for scurvy: (1) That of Wright, that scurvy is due to an acid intoxication—a condition in which there is a marked diminution in the normal alkalinity of the blood-plasma, the result of a dietary of foodstuffs which contain a large excess of mineral acids over bases—a dietary of meats, especially salted meats, and cereals, to the exclusion of green vegetables; (2) that of Liston, who ascribed the disease to infection with Ankylostoma duodenale; and (3) that of Jackson and Harley, who consider scurvy a condition of ptomain-poisoning produced through the eating of tainted animal foods. The third theory is dismissed from consideration after a short discussion, and then are detailed the results of examining 11 cases with reference to the alkalinity of the blood and the character of the food ingested, and the presence of Ankylostoma duodenale in the stools. The ovums of this parasite were not detected in any case. The facts that go to show that the patients suffering from scurvy were not affected with acid intoxication are: (1) There was no marked deficiency in the alkaline foodstuffs of the dietary before the onset of the symptoms; (2) there was no diminution in the alkalinity of the blood-serum when the symptoms were well marked; and (3) there was no improvement in the symptoms either as the result of giving a diet consisting of abundant alkaline foodstuffs or on the administration of large doses of sodium lactate. It is believed that although scorbutic symptoms may be associated with acid intoxication, other cases occur quite independently of this condition. There seems to be more than one etiologic factor and pathologic condition underlying the symptoms that are known clinically as scurvy.

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[1] Wien. klin. Rundschau, Nov. 16, 1901.

[2] Lancet, Jan. 4, 1902.

The Etiology of Scurvy.—Lewine[1] details the result of his bacteriologic investigations in 12 cases of scurvy, all of which were examined during the active febrile stage, and, in order to avoid contamination, by aseptic puncture of the liver and spleen. In 11 of the cases he obtained upon peptonized gelose a pure culture of a bacterium with rounded ends, staining very slightly, if at all, by Gram's method. He designates it Bacillus hemosepticus, and considers it identical with the bacterium that causes fowl cholera, rabbit septicemia, contagious swine pneumonia, and several other diseases, all characterized by hemorrhagic septicemia, and particularly enteritis. The difference in these cases is believed to be one of virulence for particular species of animals. Lewine regards scurvy as a chronic hemorrhagic septicemia due to a microbe which is widely distributed in nature, but does not acquire the power of causing the particular disease until it has become acclimatized in the human subject. Koch has found this organism in the mother-fluid of preserved foods, and Lewine gives details of the recent epidemic in Kazan, which is thought to have attacked mainly those that ate salt horseflesh rather than those suffering from actual starvation.

TRYPANOSOMA INFECTION.

Trypanosoma in Man.—Dutton,[2] through Ronald Ross, made the interesting and important announcement that he had discovered trypanosomas in a European resident in British Gambia, West Africa. Subsequently Dutton himself published the details of his case.[3] This at first was thought to have been the first case in which these organisms were found in man, but later it was ascertained that Forde[4] had previously observed but had not reported a case, and the report of a somewhat doubtful case observed by Nepveu[5] was found. The characteristics of the disease in Dutton's patient were its chronic course; the general wasting and weakness; irregular rises of temperature, never very high and of a relapsing type; local edemas, especially of the eyelids, face, and legs; congested areas of the skin; enlargement of the spleen; and constant increased frequency of the pulse and respiration. There was no organic disease of the heart or kidneys, and no malarial parasites in the blood on repeated examinations.

Trypanosoma is a genus of flagellated protozoa very closely related to Trichomonas, but while the latter are more or less harmless parasites, the pathogenicity of the former is well established.[1] Nearly all species of trypanosoma thus far described are parasites in the blood, while the few species reported for the intestine probably are not correctly classified in this genus. Trypanosoma lewisi, a species which infests rats, is the best-known form, and is not especially pathogenic; it is transmitted from rat to rat by fleas and lice. It is Trypanosoma evansi which infests horses, causing a pernicious anemia known as "surra." This disease, which is carried by gad-flies (Tabanus), is invariably fatal to horses, mules, and camels in India, and has recently been reported as causing great losses among United States cavalry horses in the Philippines, a fact that led the Secretary of Agriculture to prohibit the landing of any animals from the Philippines at any port of the United States. Tsetse-fly disease of Africa, which Livingston and other travelers describe as so fatal to horses, cattle, and other stock, is caused by Tr. brocei and is transmitted by the noted tsetse-fly (Glossina morsitans). Mal de caderas is an invariably fatal disease among horses in South America, and is caused by Tr. equinum; its mode of transmission is unknown. Tr. equiperdum (Tr. rougeti) is looked upon as the cause of dourine (gonorrhea) in horses in France, Spain, and Algiers, a disease which is transmissible by coitus. Other species of Trypanosoma (Tr. avium and Tr. eberthi) are reported for birds, the former in the blood, the latter in the intestine. Tr. rotatorium is the type of the genus and occurs in frogs; still other species (Tr. cobitis, Tr. carassii, Tr. remaki, Tr. soleæ) are described for fish; Trypanosoma danilewskyi (Trypanoplasma danilewskyi) was described for the intestine of leeches in France, but is probably a parasite of some vertebrate upon which the leech had fed. Tr. balbianii of oysters is probably not a true Trypanosoma. These organisms are minute mononuclear, unicellular structures, several times as large as a red blood-corpuscle, elongate, more or less fusiform, and provided with a lateral, longitudinal, undulating membrane somewhat similar to the membrane of Trichomonas vaginalis of the human vagina. The free margin of the membrane is formed by a thickened structure which in reality represents a flagellum; this begins near the posterior end of the so-called centrosome and extends along the margin of the membrane to the anterior end of the body, where it becomes a true flagellum. The reproduction is nonsexual by longitudinal division, and no sexual stage is known for it corresponding to the sexual stage of Plasmodium malariæ in the mosquito. The parasites live in the serum, attacking and shaking the red blood-corpuscles much as a dog shakes a rat. This naturally results in great decrease of the red cells, so that an anemic condition results. Trypanosomatic diseases are wet-weather maladies, and are somewhat similar to malaria. There is a general though not absolute periodicity, the temperature rising with the increase of the parasites, then falling more or less suddenly to normal or subnormal, the hematozoa at the same time disappearing more or less completely from the circulation. Aside from the anemia and progressive emaciation, the most marked symptoms are uncertain gait (sometimes amounting to a paralysis), ravenous appetite, extreme thirst, highly colored urine, offensive feces, urticaria, edema, petechias on the mucous membranes, jaundice, and violent heart action. On necropsy the spleen is found enlarged and there are ulcers in the stomach; aside from these there are no specific trypanosomatic lesions. Treatment has been unsatisfactory, but some good results are reported from the use of arsenic. From a military and economic standpoint, trypanosomatic diseases are exceedingly important, and as some species of Trypanosoma—for instance Tr. equinum—are transmissible to monkeys; further, since several of the species are infectious for widely distinct zoologic genera, it is not improbable that they may infect also man.

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[1] Arch. des Sci. Biol. de St. Petersbourg, No. 3, 1901; Brit. Med. Jour., Nov. 2, 1901.

[2] Brit. Med. Jour., I, 42, 100, 1902.

[3] Brit. Med. Jour., II, 881, 1902.

[4] Jour. of Trop. Med., Sept. 1, 1902.

[5] Compt.-Rend. de sci. de la Soc. de Biol., Dec., 1898.

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[1] Amer. Med., III, 240, 1902.

A New Form of Trypanosoma.—Trypanosoma theileri is mentioned by Bruce.[1]

DYSENTERY.

Epidemic Dysentery in Japan.—Shiga[2] gives the following as his conclusions from his continued studies of epidemic dysentery in Japan: (1) Bacillus dysenteriæ is a constant occurrence in all cases of dysentery; (2) it occurs only in cases of dysentery, never in healthy individuals or in patients suffering from other diseases; (3) the occurrence of the bacillus in the dejecta is a concomitant of the morbid process; (4) the bacillus is found almost entirely in the deeper layers of the intestinal wall; (5) the bacillus or its toxins have a tendency to produce hemorrhage; (6) the bacillus of dysentery exhibits agglutination-phenomena only with the blood of patients suffering from dysentery, not with the blood of healthy individuals or patients suffering from other diseases; (7) this faculty of producing agglutination varies according to the stage of the disease, being rapidly developed and reaching its highest point just before convalescence; (8) dead cultures of the bacillus, when injected subcutaneously into healthy individuals, produce marked local inflammatory infiltrations, the reaction being considerably lessened in virulence in the case of patients recovering from the disease; (9) Pfeiffer's reaction of the dysentery bacillus is especially marked during the convalescence of the patient; (10) the immune-serum possesses both prophylactic and therapeutic action in combating the disease.

Dysentery in Children.—Amberg[3] has been able to collect 6 cases of the disease in American literature, adding 5 to the number. The ages of the patients varied between 2¾ years and 5 years. The mild type of the disease, the usual one in children, was noticed by Amberg in 4 of his cases, the fifth being moderately severe. Two of the patients had prolapsed rectum; none of them had hepatic complications. The diagnosis in all cases was based upon the finding of motile amebas containing red corpuscles in the feces. Charcot-Leyden crystals, eosinophile cells, and free granules were present in 4 cases and Amberg emphasizes the fact that should Charcot-Leyden crystals be found in the stools of a child, amebic dysentery should always be thought of, especially as the amebas are found only on repeated examinations.

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[1] Brit. Med. Jour., April 12, 1902; Epitome, No. 237.
[2] Deut. med. Woch., Nov. 7, 1901.
[3] Bull. Johns Hopkins Hosp., Dec., 1901.

MISCELLANEOUS INFECTIONS.

The Pathogenic Significance of Balantidium coli.—Henschen[1] reports two additional cases (making a total of 8 cases) of chronic diarrhea due to Balantidium coli. In 5 of the cases the destruction of the parasites was followed by rapid recovery of the patients. The treatment advocated consists in the use of large enemas (2500 cc.) to which are added 50 to 75 cc. of vinegar and 5 to 7.5 grams of tannin. A fatal case of diarrhea originating in the Philippines and revealing great numbers of Balantidium coli in the stools is reported by Strong and Musgrave.[2] An eosinophilia was present during life. At the necropsy the small and the large intestine were congested and covered with considerable mucus, and the large intestine revealed, in addition, a number of shallow ulcers. Balantidium coli was found in the mucosa, even in the submucosa and muscularis. Solowjew[3] has studied the subject exhaustively and reviewed the literature. He maintains the pathogenicity of the organism, showing how it penetrates the mucosa and submucosa and infiltrates the muscularis, giving rise to necrosis and ulceration. He believes that the presence of the parasite in the depths of the intestinal wall explains not only the development of the disease, but also the many relapses that follow after apparent destruction of the parasites.

Pneumonia of Measles.—According to Steinhaus,[4] the pneumonia of measles is at its beginning neither a lobular nor a catarrhal inflammation; the foci are very small, several of them occurring in one lobule, and each focus contains fibrin. The foci through their confluence may produce a lobular and even a lobar consolidation. The primary lesion is a bronchiolitis and a peribronchiolitis, the inflammation being propagated along the course of the lymphatic vessels. Some of the foci arise acutely, and are associated with exudation into the alveoli, and the formation of fibrin; the process may spread to neighboring alveoli or ascend along the originally affected alveolus. Others of the foci are more chronic; they are not associated with alveolar exudation, but rather with proliferative changes in the interstitial tissue. Interstitial inflammations occur much earlier in the pneumonia of measles than in croupous pneumonia. Leukocytes infiltrate the surrounding tissue in the case of the acutely arising foci, lymphocytes in the more chronic inflammations. Tuberculosis is a frequent sequel after the pneumonia of measles, and is usually secondary to a tuberculosis of the bronchial lymph-glands.

Leptothrix Infections in Man.—Pearce[5] reports 2 unique cases of leptothrix infections in man, one a case of leptothricial necrosis of a part of the cartilage of the larynx, the second a case of leptothricial cholelithiasis and choleangitis. In the first of the cases there was tuberculosis of the lungs, the intestines, the mesenteric lymph-nodes, the prostate gland, left seminal vesicle, vas deferens, and epididymis; tuberculosis of the left middle ear with involvement of petrous and mastoid portions of temporal bone, and extension to the dura mater, fatty degeneration of heart, liver, and kidneys. The larynx presented 3 different processes, tuberculous, secondary to that of the lungs; a nontubercular abscesss of the submucosa, rupturing into the cavity of the larynx, probably pyococcal in origin; an invasion of the posterior wall of the abscess by streptothrix, resulting in the disorganization of the cartilage of the larynx, and involvement and perforation of the esophagus. In the second case there were found gall-stones in the diverticulum of Vater and hepatic duct, cholangitis, slight cirrhosis of the liver, chronic diffuse nephritis, with fibroma of kidney, chronic gastritis, and arteriosclerosis. Leptothrix was the only microorganism in the concretions, and with the colon bacillus was found in large numbers in the bile. Histologic descriptions of the organs are given.

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[1] Arch f. Verdauungskrankheit., VII, 501, 1901.
[2] Bull. Johns Hopkins Hosp., XII, 31, 1901.
[3] Centralbl. f. Bakteriologie, XXIX, 821, 849, 1901.
[4] Zeigler's Beitr. zur path. Anat., etc., 1901, vol. XXIX, p. 524.
[5] Univ. of Penna. Med. Bull., Aug., 1901, p. 217

A New Acid-proof Streptothrix Pathogenic for Man and Animals.—Birt and Leishman[1] have isolated a new streptothrix from a male of 26, suffering from hectic fever, right-sided pleural effusion, enlargement of the liver, and hiccup, with the expectoration of a mucopurulent material. In the sputum and in the pleural effusion the organism was found. At the autopsy the right pleural cavity was found to be the seat of advanced empyema, and the right lung was in the condition of chronic bronchopneumonia, but was free from nodules and cavities. The left lung was studded throughout with small cirrhotic nodules, showing no caseation and differing in many respects from miliary tubercles. From the nodules on the left lung, the empyema-pus, and the pericardial fluid, the streptothrix was obtained; in pure culture from the pericardial fluid, and in conjunction with pyogenic organisms from the pus and the lung. The organisms grow very slightly, if at all, at room-temperature. They grow slowly on gelatin, without producing liquefaction. On agar their growth is rapid, in the form of a snow-white, dry, powdery material, taking on a pale coral-pink tinge after a few days. They also grow in bouillon, and on potato—on which the growth looks like a splash of plaster-of-paris. There is no growth on blood-serum. The organism is aerobic and almost thermophilic. It occurs in two forms: as a branching network of mycelial threads, and in the so-called streptococcic form. It stains by Gabbet's method. Like all streptothrices, it is nonmotile. It is pathogenic for guinea-pigs, causing their death in from 5 to 6 weeks, large collections of caseous material being found gluing together the omentum and small intestine.

Staphylococcus Pyemia.—Muscatello and Ottaviano,[2] as a result of considerable experimental and other study, conclude that the three principal forms under which staphylococcus general infection occurs—

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[1] Jour. of Hyg., 1902, vol. II, No. 2. [2] Virchow's Archiv, CLXVI, 212, 1901.

toxemia, toxemia with bacteremia, and pyemia—are intimately related to one another. The development of the one or the other manifestation of the general infection, all of which result from the entrance into the circulation from a local focus of toxins and cocci, is dependent upon (*a*) the degree and nature of the virulence of the cocci (specific virulence); (*b*) the presence or absence of local predisposing factors (*locus minoris resistentiæ*); (*c*) the greater or less absorptive powers of the part first involved; (*d*) the degree of the general and local natural immunity of the affected individual. The first, the constant, and the basic manifestation of all forms of general infection caused by staphylococci is the intoxication. This is the direct cause of the diffuse alterations in the organs, and it prepares the tissues for the metastatic localization of bacteria; for the diffuse alterations not only seriously compromise the general functions of the body, but they also give rise to abnormal local physical conditions (*locus minoris resistentiæ*) that favor the localization of cocci and the production of focal lesions. For the production of a pure bacterial pyemia the following conditions are requisite: (*a*) moderate intoxication which reduces to a certain extent, but does not abolish, the powers of resisting of the body; (*b*) a *locus minoris resistentiæ* (degenerations, retarded secretions or circulation, severe local lesions, hemorrhages, etc.); (*c*) the concurrent entrance of many cocci into the circulation. The entrance of few or of many cocci into the circulation is dependent in part upon the local powers of absorption, in part upon the virulence of the cocci. In the pure bacterial pyemic form of general infection the metastatic foci are related to the physical conditions associated with local lessening of resistance. The metastatic foci in the kidneys are in large part abscesses the result of excretion. The foci in the voluntary muscles and the heart are related to degenerations, necroses, and circumscribed hemorrhages. The foci in the liver and the lungs are very probably related to alterations produced in the blood-vessel walls by the intoxication.

The Identity of the Streptococci Pathogenic for Man.—In spite of the numerous morphologic differences which are noted in streptococci derived from different diseases, Marmorek[1] is of the opinion that they belong to one and the same class, and that the only thing necessary to overcome these differences, or to produce others, is a slight change in the culture-medium. The proof of their identity lies in the fact that they all possess 3 peculiarities, which he has found in 42 specimens coming from different diseases: (1) The red blood-cells of rabbit's blood are dissolved by them *in vivo:* the more virulent the culture, the more rapidly and completely is this done; (2) they cannot grow in a culture made of their own filtrate; other organisms, as the pneumococcus, can grow in it, but to produce a further growth of the streptococci fresh bouillon or other mediums must be added; (3) the immunization of animals by the use of antistreptococcic serum. The virulence of all streptococci may be increased *ad libitum*. The streptococcus of scarlet fever possesses quantitative but not qualitative differences.

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[1] Berl. klin. Woch., April 7, 1902.

The Bacteriology of the Venereal Soft Sore.—Von Zeissl[1] analyzes the researches that have established a probability that approaches a certainty that Ducrey's bacillus is the specific cause of the venereal soft sore. Before the days of bacteriologic investigation the soft sore was believed by many to be due to a specific cause. In 1887 Welander described a bacillus in the discharge which he considered characteristic. Ducrey, in 1889, cultivated an organism on its natural medium, the human skin; he obtained secretion for inoculation from 3 typical venereal ulcers, and carried out 3 series of inoculations up to the twenty-fifth generation. In the pus from the original ulcers were a variety of organisms, but none constant. In the artificial pustules their number decreased, until at the fifth or sixth generation a product was obtained which was in the highest degree virulent, but from which no cultures were obtained on ordinary mediums. In this product a certain bacillus was constantly found with the microscope; inoculation on animals was without effect. Later Krefting demonstrated the same bacillus in the secretion from the ulcers, in the pus of buboes, in sections of extirpated sores, and in the walls of buboes. Unna obtained similar results, and many others confirmed them. Istamoff and Aspianz, in 1897, obtained positive culture results on an agar medium made from macerated human skin. Lenglet also cultivated on a similar medium, the ulcer being first disinfected with sublimate to destroy the ordinary pus organisms. Besançon and others have obtained pure cultures on the following mediums: 2 parts of agar melted at 50° C. and mixed with 1 part of blood either from the human subject, the dog, or the rabbit. Inoculations of the organism on the human subject were positive. That it is the specific cause of venereal soft sores is shown (1) by the constancy of its presence; (2) by its exclusive presence in the later generations of the artificially produced sores; (3) by their inclusion in leukocytes in smear preparations and sections; (4) by their presence in certain cases in buboes following the sores; (5) by their absence from other similar ulcerative processes; (6) by the fact that pure cultures produce typical venereal sores in the human subject. It has been shown that the pus of many unopened buboes contains no organisms. Strauss believed that all those giving positive results had been inoculated after opening with the discharge from the sores; but although the pus in these abscesses may be free from organisms, it has been shown by Audry and others that the specific streptobacillus is to be found in the abscess-walls in many cases, and in inflamed glands which have not reached the abscess stage. It would appear from a general survey that there are buboes in which the pus is free from microorganisms, others in which it contains staphylococci, and others in which it contains Ducrey's bacillus. Now that the difficulties of culture have been overcome this question of the infectivity of buboes can be settled.

The Pathogenicity of Bacillus Pyocyaneus.—Sottman[2] reports the case of a boy of 13 who was taken ill suddenly with high irregular fever and grave general symptoms pointing to a rapidly developing septic pneumonia. In the course of it, meningeal symptoms made their appearance, with occasional diarrhea. He died on the eighth day. The postmortem examination showed serofibrinous pleurisy; inflammation of the greater part of both lungs with partial necrosis, manifesting itself in the form of yellowish and grayish-white foci; congestion and cloudy swelling of spleen, kidney, liver, pancreas, etc., necrotic inflammation of the gastric and intestinal mucosa. Bacillus pyocyaneus were procured in almost pure culture from all the affected organs. Sections of the lung showed, in addition to the characteristic picture of pneumonia, these organisms and numerous dust-cells. He considers these cells to have been the carriers of the infection, and the lung to have been the primary seat of the disease. The intestinal tract was affected secondarily. This case proves that Bacillus pyocyaneus is not always a harmless saprophyte; to be pathogenic it need not be associated with other organisms; and that to be affected by it, it is not necessary to be in low health.

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[1] Centralbl. f. Bakt., Feb. 26, 1902; Brit. Med. Jour., 1902, I.
[2] Deut. Arch. f. klin. Med., 1902, LXXIII, 650.

Bacillus Anthracoides.—Bainbridge[1] describes an organism that he terms Bacillus anthracoides isolated from a bundle of Chinese horsehair which was being examined for—and actually contained—anthrax spores. The two bacilli resemble each other in size, shape, capsule, staining reactions, and growth on agar. On the other hand, Bacillus anthracoides differs from anthrax in its motility, growth on gelatin, more rapid growth on most mediums, and especially in its very limited pathogenic power.

Noma.—Recently Sailer[2] found the diphtheria bacillus in two cases of noma complicating typhoid fever. The most recent observation upon the subject is that of Trambusti,[3] who, in a case of spontaneous noma involving the nose, lip, and jaw, found a hitherto undescribed organism. In form, it was a short bacillus with rounded ends, not motile, and staining by Gram. It grew upon all mediums; best at the body-temperature. It was pathogenic for laboratory animals. By passage through animals its virulence could be so enhanced that it killed a rabbit weighing 1.5 kilos in less than 7 hours. Injected alone, either subcutaneously, intraperitoneally, or intravenously, it caused death by septicemia, without producing gangrene. When, however, it was introduced into the mucous membrane of the mouth, where it came into contact with the common bacteria of that cavity, there was distinct gangrenous necrosis. [Trambusti's communication is too brief to permit a definite judgment to be formed as to the nature of the organism discovered by him. That the diphtheria bacillus, however, bears a distinct relation to many cases of noma has been proved by the beneficial effects of antitoxin injections. Even in the absence of a bacteriologic examination, in cases of cancrum oris and of noma elsewhere, it is advisable to use injections of diphtheria antitoxin.]

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[1] Jour. of Path. and Bact., VIII, 117, 1902.
[2] Proc. Path. Soc. of Phila., N. S., vol. III, Nov., 1901.
[3] Il Policlinico, vol. IX, Jan., 1902.

The Spread of Leprosy and the Fish Hypothesis.—Neve,[1] discussing leprosy, gives some observations concerning the hypothesis of its spread by means of fish as applied by Kashmir. It is believed that fish is not an actual cause *de novo* of leprosy, but that it, as well as other foodstuffs, may serve as a carrier of leprosy virus with which it may have been in accidental contact.

The Diagnosis of Glanders by the Strauss Method.—Frothingham[2] points out that Strauss[3] was the first to demonstrate that if virulent glanders bacilli are introduced into the abdominal cavity of the male guinea-pig peculiar suppurative lesions of the scrotal peritoneum generally resulted in a few days—a method of investigation since known as the Strauss method. Frothingham's investigations comprise positive results in the cases of 123 horses and 2 men, the inoculating swab having been taken from the nose in 102 of the cases and from the skin in 23 of the cases. Positive results were obtained at the first test in 105 cases, at the second test in 16 cases, at the third test in 2 cases, and at the fourth test in 2 cases. Scrotal lesions were observed on the second day after the inoculation in 59 cases, on the third day in 51 cases, on the fourth day in 14 cases, and on the fifth day in 1 case. Negative results were obtained in the cases of 189 horses, in 35 of which the horse, afterward being killed, was found to be glandered. The number of failures is thought not to be remarkable, in view of the undoubted occurrence of differences in the virulence of bacteria, of a poor or insufficient discharge, and of the absence of bacilli in the very early stages. Of the 35 cases, 29 were tested but once; and of these 29, 25 were condemned at a later inspection, 3 were condemned at the time the swab was taken, and 1 died of glanders before the second inspection. There were, therefore, but 4 absolute failures. Other important points to be noted are that when glanders lesions are healing, the bacilli in them are probably less numerous and perhaps less virulent, and that the guinea-pig is not highly susceptible to glanders—whence it might be advisable to use field-mice or other susceptible animals. The Strauss method is believed to be the most valuable at our command.

The Cattle Plague in Shanghai and its Limitation by the Gall Immunization of Koch.—According to Arthur Stanley,[4] it is possible to produce immunity against rinderpest in the following ways: (1) By the subcutaneous injection of bile from an animal dead of cattle plague (Koch); (2) by the injection of the blood of an animal which has had cattle plague; (3) by the injection of the blood of an animal which has had cattle plague, together with the blood of an animal which has cattle plague (simultaneous method of Kolle and Turner); (4) by a previous attack of the disease. The first two represent passive, and the second two active, immunity. The author found the first method to be the most feasible, inasmuch as it is inexpensive and easily applicable when once a case of cattle plague has furnished the bile. The dose used was 10 cc. of the bile of an animal dead of the plague.

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[1] Brit. Med. Jour., May 3, 1902. [2] Jour. of Med. Research, vi, 331, 1901.

[3] Compt.-rend. Acad. des. sci., cviii, 530; Revue Vetern., June, 1889.

[4] Jour. of Hyg., vol. ii, No. 1, 1902, p. 43.

Bacillus Coli Communis in Dough, Flour, and Grain.—Papasotiriu,[1] as well as other observers, has found Bacillus coli communis constantly present in flour; often in grain, such as wheat, oats, rye, barley, and maize; and in peas and beans. He is of the opinion that it is the cause of the working of leaven. It can also be frequently found in water, and its presence there in small numbers is of no diagnostic importance; large numbers in fresh water raise the suspicion of fecal contamination, but it should be remembered that the waste water from bakeries may carry large quantities of the bacillus coli to the water. The bacillus propagates in water under favorable conditions, such as high temperature and the presence of carbohydrates.

A New Gas-producing Bacillus.—Uffenheimer[2] reports a case of general septicemia following childbirth, which was characterized by general gas-production in addition to the presence of pus-producing microorganisms. The gas bacillus was isolated from all the organs of the body. It differs morphologically and culturally from all the species thus far described. On account of its resemblance to Welch's Bacillus aerogenes capsulatus, he calls it Bacillus aerogenes aerophilis agilis. It is facultatively aerobic, excessively motile, produces gas, and, under certain conditions, spores.

The Identification of the Colon Bacillus by Reaction Produced in Culture-mediums Containing Neutral Red.—To determine if Bacillus coli communis can be differentiated from other bacteria, and especially from Bacillus typhosus, by using culture-mediums containing neutral red, Rosenberger[3] performed a large number of experiments. He concludes that while not affording a specific reaction in the case of Bacillus coli communis, neutral red agar should be classed as a valuable differentiating medium. Furthermore, the test-medium should not be depended upon as the only differentiating factor in examining water, as several very common bacteria found in water give the same reaction.

A Rapid Method of Detecting Bacillus Coli Communis in Water.—Stone[4] describes a rapid method of detecting Bacillus coli communis in water. It requires only 72 hours to be sure of the presence of the organism, while specimens not containing glucose-fermenting bacteria can be declared negative in 24 hours. To estimate the total number of bacteria to the cubic centimeter, the bottle containing the water is thoroughly agitated, a 1 % solution of it made with distilled, sterilized water, the mixture well shaken, and 1 cc. of this mixed with a tube of plating agar. This is permitted to solidify in a Petri dish, and grown at 22° C. for 96 hours. The number of colonies, multiplied by 100, represents approximately the number of individuals to the cubic centimeter of the original water. To determine the presence of Bacillus coli communis: (*A*) A Smith's tube filled with a 2 % glucose bouillon is inoculated with 1 cc. of water to be examined and grown 24 hours at 38° C. If no gas is formed, the absence of the colon bacillus is shown. (*B*) If from 25 % to 70 % of gas is formed in the closed arm, a tube containing 10 cc. of neutral broth to which has been added 0.3 cc. Parietti's solution is inoculated with 0.5 cc. of the contents of A and grown 24 hours at 38° C. (*C*) A second Smith's tube is inoculated with 0.5 cc. of the contents of B and grown 24 hours at 33° C. If there is no gas, we may be sure the gas-producer in A was not the colon bacillus. If, on the other hand, gas is produced in this tube, we may be reasonably sure that Bacillus coli communis is present. (*D*) Further confirmation may be obtained by ascertaining the gas formula from C. The formula for the colon group is $H / CO_2\text{-}2 / 1$, further. (*E*) A pure culture can be easily obtained from B by plating and the following reactions obtained: (1) Gelatin stab does not liquefy after 7 days at 22° C. (2) Litmus milk reddens and coagulates in 24 hours at 37°. (3) Dunham's solution: indol formed in 3 days at 37° C. (4) Morphology in bouillon: sluggishly motile (?) bacillus. To estimate the number of Bacilli coli communis, a litmus lactose of agar plate of 0.5 cc. of the original water is made. After solidifying, this plate is inverted and grown 24 hours at 38° C., when the colonies of Bacillus coli communis may be identified as red colonies on a blue plate.

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[1] Arch. f. Hyg., Bd. XLI, Heft 3, 1902.
[2] Ziegler's Beitr. zur path. Anat., etc., 1902, vol. XXXI, p. 383.
[3] Phila. Med. Jour., Mar. 8, 1902.
[4] Amer. Med., Jan. 25, 1902.

The Effects of Change of Color upon Pigment Bacteria.—Oliver[1] undertook an experimental study of the effects of change of color on pigment bacteria, the investigations consisting of determining the effects produced on bacteria that had received their designative names from their characteristic hues by light that was caused to pass through transparent sheets of definitely gaged colored glass. His conclusions are as follows: (1) Color changes, both of kind and intensity, take place in and around many chromogenic bacteria when such microorganisms are placed under different color conditions; (2) rehabilitation of chromogenic bacteria into old color environments after having later obtained a new color value under any particular color condition is quite frequently accompanied, sooner or later, with a return of the germ's color equivalent to that which it primarily held while in its original situation; (3) differences of color conditions in pigment most probably signify in part relative changes in the various methods of obtaining sustenance, peculiarities in the kinds and the ratios of foodstuffs, and irregularities in the character of resulting excreta; each species of color bacterium exhibiting its chromogenic change in a typical and relevant manner, a behavior that has its determining effect upon the very life of the germ itself; (4) the naming of bacteria by specific coloration is of value only when the actual habitat of the microorganism is understood; and (5) as a postulate conclusion based upon these facts it is most certain that all living faunal and floral color-changes of true objective type are expressions of biochemic peculiarities taking place in and around such organisms—a mere difference of molecular motion, as it were, in part dependent upon the relationship existing between the active life force and coexisting conditions.

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[1] Am. Jour. Med. Sci., cxxiii, 647, 1902.

Flagella Staining with Night-blue.—Letchworth Smith[1] describes the following method of staining flagella devised by George H. Newman: (1) Make an emulsion of a fresh bouillon culture of the bacterium by adding one or two loopfuls to a small quantity of distilled water. (2) Transfer a loopful of the emulsion to a clean, dry slide or cover-glass, spread very gently, and allow to dry in the air. (3) Though special fixation is unnecessary, it is better to expose the cover-glass to a mild degree of heat—hold in the fingers and dry thoroughly rather than heat. (4) Filter the stain on the dry film and leave in contact for 5 or 10 minutes. The stain consists of tannic acid 1, potassium alum 1, and distilled water 40. Dissolve by a few minutes' shaking or leave in the incubator overnight. Dissolve night-blue 0.5 in alcohol (95 % or absolute) 20. Mix the two solutions thoroughly and remove the heavy precipitate by filtering. The stain acts better when a few days old. (5) Wash carefully but thoroughly in water. (6) Apply a saturated aqueous solution of gentian-violet for about 2 minutes—to stain the bodies of the bacilli. (7) Wash in water, dry with smooth blotting-paper, and mount in balsam. Should one desire to obtain a background as free as possible from precipitate, as for photographic purposes, it is better to apply the night-blue solution to the slide inverted.

The Relation of Insects to the Spread of Disease.—McFarland[2] summarizes the ways in which insects may become carriers of disease: (1) They may carry from place to place disease-producing microorganisms which have collected upon their bodies and limbs, and occasion infection directly by alighting upon wounds, and indirectly by alighting upon foodstuffs. The common house-fly, in particular, belongs in this category. (2) They may carry within their bodies germs of disease which have entered with their food, and which may subsequently be deposited elsewhere with their feces. The feces of flies which were fed upon sputum have been found to contain tubercle bacilli. (3) Suctorial insects, by taking blood containing parasites from the bodies of diseased animals, may carry these upon their probosces into the next animal bitten, directly and immediately infecting it. Anthrax, plague, and trypanosoma have been spread in this manner. (4) They may take infectious germs into their bodies and transmit them to their offspring, whose bites are infectious; this is seen in the Texas fever of cattle. (5) They may take into their bodies parasitic organisms which there undergo a further development, the insects acting either as an intermediate or a definitive host, and transmitting the parasite to other animals in some changed form in which they are infective. Malaria, yellow fever, and filariasis may be thus transmitted. (6) The insect may become infected with pathogenic organisms, die, fall into foodstuffs, and thus impart infection to man. Plague and other infection may be thus transmitted.

Infection Spread by Domestic Pets.—Meany[3] discusses the relations of human diphtheria to diseases of the lower animals and mentions several outbreaks of the disease which have been traced to pigeons, chickens, and cats; he also recalls an outbreak of pneumonic plague on a vessel, which was traced to a cat. He closes his remarks by warning against too early lifting of quarantine in infectious diseases, as the diseases are often kept alive by convalescents associating with the well; disinfection should be carried on much more strictly than is the custom.

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[1] Jour. of Med. Research, vi, 341, 1901.
[2] Medicine, viii, p. 1, 1902.
[3] N. Y. Med. Jour., Aug. 17, 1901.

The Significance of the Recent American Cases of Hookworm Diseases (Uncinariasis or Anchylostomiasis) in Man.—Stiles,[1] reviewing the recent American cases (35 since 1893), points out that uncinariasis or anchylostomiasis is a specific zooparasitic disease caused by hookworms which inhabit the small intestine. Its chief symptoms are: Anemia, with the circulatory symptoms found in all extreme anemias—namely, dizziness, palpitation, hemic murmurs, colicky pains in the abdomen; great weakness, in some cases with considerable emaciation; constipation or diarrhea, stools sometimes brownish or bloody; nausea; edema. The only positive diagnosis is by finding the parasite or its eggs in the stools. It may affect any class of patients, but is more frequent in persons whose daily life brings them in contact with damp earth (miners, brickmakers, excavators, dirt-eaters, etc.). The parasite causing uncinariasis is a worm belonging to the nematode family Strongylidæ, species Uncinaria duodenalis. There are many uncinariæ, but the latter is the only species of the genus which has been found in man, and man is the only host in which it has been satisfactorily demonstrated. The following is its life-history: The eggs are laid in the intestinal tract of the patient by the female worms, and are discharged in the feces. They will not develop into adult worms in the intestine, but must first pass out of the body. A short time after escaping in the feces each egg develops a minute embryo, which is known as a rhabditiform embryo. The embryo of the hookworm (uncinaria) lives in water or in moist ground. In its evolution the worm casts its skin 4 times, thus passing through 5 stages, and changes its structure so as to assume more and more the characters of the adult. During these changes the sexes become differentiated. Some of these changes occur in water or in moist ground, and the rest after infection takes place. About the second or third day the embryo casts its first skin. After the fifth day the young worms begin to show signs of a second ecdysis, at the same time they become more motile, and contrast from their outer skin, thus forming the stage which has been described as an encystation. Upon being swallowed these young worms undergo further ecdyses, changing their internal organization at the same time. We may recognize a third stage without buccal capsule; a fourth stage, with a provisional buccal capsule; finally, a fifth stage, with the definite buccal capsule, corresponding to the adult form. The worm in the fifth stage is about 1.9 (male) to 2 mm. (female) long, 12 to 14 μ in diameter—very much smaller than the adult forms, which are from 7 to 9 mm. (male) and 9 to 11 mm. (female) long. It is estimated that the parasites require about 4 to 5 or 6 weeks from the time of infection to become mature. The worms are swallowed in drinking-water or in contaminated food, during or after the second ecdysis.

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[1] U. S. Dept. of Agriculture, Bureau of Animal Industry, Eighteenth Annual Report, p. 183, 1901.

A New Species of Hookworm (Uncinaria americana) Parasitic in Man.—While studying hookworms, Stiles[1] accidentally discovered a new variety of uncinaria; he calls it Uncinaria americana, and describes it as follows: Specific diagnosis—Differs from Uncinaria duodenalis chiefly in the following characteristics: Ventral recurved hook-like teeth are absent from the mouth, their place being taken by a pair of semilunar plates, somewhat similar to Uncinaria stenocephala. Dorsal conical tooth projects prominently into the buccal capsule. Male: Dorsal ray of caudal bursa divided to its base, each branch being bipartite at its tip. Female: Vulva in anterior half of body, but near the equator. Eggs: Slightly larger, 64 to 72 μ by 36 to 40 μ, in some cases partially segmented, in others containing fully developed embryo when oviposited. Type host—Man (Homo sapiens).

Pathologic Report upon a Fatal Case of Enteritis with Anemia Caused by Uncinaria duodenalis.—Yates[2] makes a pathologic report upon a fatal case of enteritis with anemia caused by Uncinaria duodenalis. The patient, an English sailor, had been feeling bad for the past 6 months; he complained of loss of appetite, failing digestion, colicky abdominal pain, and some diarrhea, all of which had gradually become worse. Of late his stools had all contained a great deal of blood. Physical examination revealed nothing except pronounced anemia, poor general condition, dulled mentality, and some abdominal tenderness. At the autopsy were found catarrhal gastroenteritis, with hemorrhages due to Uncinaria duodenalis; a few adult worms were found in the duodenum; the jejunum and ileum contained thousands of them in living condition, with their heads buried in the mucosa. None were found in the last few centimeters of the ileum.

INTOXICATIONS, IMMUNITY, HEMOLYSIS, ETC.

Inherited Immunity.—Dzierjgowski,[3] with a view to demonstrate the truth or falsity of his belief that immunity transmitted to offspring is not really hereditary but acquired by the ovum or fetus in the body of the mother, studied in hens' eggs the immunity to diphtheria toxin. He found antitoxin demonstrable only in the yolk of the egg, and proved that it is not destroyed by any enzymes making their appearance during the development of the egg, but penetrates into the fetus. In the latter it is confined for the most part to the serum of the blood. In the yolk the antitoxic principles are for the most part localized in the formative portion. The antitoxin can be extracted from the yolk by water and separated by dialysis; it passes unchanged through the Chamberlain filter and does not contain phosphorus, and therefore does not belong to Hoppe-Seyler's group of vitelline substances. In all probability it is a globulin, and identical with the substances of globulin nature described by Gross as existing in the yolk. Dzierjgowski arrives at the following conclusions: The albuminoid substances contained in the yolk and endowed with antitoxic properties correspond in character with those of the blood-serum. As the antitoxin of the blood can pass into the yolk unchanged, so it can pass from the yolk to fetus without modification; it would appear from this that inherited immunity does not depend on a capacity acquired by the embryonic cells to elaborate antitoxin, but simply on the fact that antitoxin passes from the maternal blood to the fetus. In other words, natural immunity is not an active immunity transmitted by the mother and showing itself in a capacity of the cells to manufacture antitoxin, but it is a passive immunity determined by the amount of antitoxin which has passed into the fetus, and which has nothing to do with any activity of the cellular elements of the offspring. A thorough **review of current theories regarding immunity** is given by Ritchie.[1]

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[1] Amer. Med., May 10, 1902, p. 777.
[2] Johns Hopkins Hosp. Bull., Dec., 1901, p. 366.
[3] Arch. d. Sci. Biol., VIII, 5; Brit. Med. Jour., May 10, 1902.

Methods of Conducting Hemolytic Experiments.—Petrie,[2] pointing out that hemolytic tests present a wide margin of possible fallacy in the conclusions arrived at from positive results, advocates the following steps in performing the tests: (1) Use absolutely fresh unclotted blood; (2) prepare in test-tubes of equal size known percentages of the hemolysin in isotonic oxalate solution; (3) add to each of the tubes exactly the same amount of blood and mix well; (4) incubate tubes for the same length of time at 37° C.; (5) centrifugalize till all the corpuscles settle in the bottom of the tube, forming a sharp line of demarcation between the blood and the supernatant liquid; (6) in every experiment have control-tubes containing isotonic salt solution alone; and (7) in doubtful cases examine microscopically.

Leukolytic Serum in a Case of Lymphatic Leukemia.—Franke[3] presents a short preliminary note on a leukolytic serum that he obtained from a case of lymphatic leukemia.

The Presence and Formation of Agglutinins in the Blood.—Ruffer and Crendiropoulo,[4] from the result of their studies, express the opinion that cultures of a microbe freed from that microbe by infiltration, dialysis, or centrifugalization, have a distinct though feeble agglutinating effect on that particular microbe. The age of the microbe and the constitution of the medium are important factors in determining the quantity of agglutinins present in such cultures. The red blood-corpuscles of nonimmunized and of immunized animals contain no trace of agglutinins; on the other hand, the polynuclear leukocytes of nonimmunized animals always possess an agglutinating power greater than, or more rarely equal to, that of the serum. They may, therefore, be rightly considered as the producers, or at any rate the carriers, of the agglutinins. In immunized animals the specific agglutinins appear in the polynuclear leukocytes, and are therefore probably formed in them. The quantity of agglutinins begins to increase 30 to 48 hours after the injection, and goes on increasing up to the tenth day or thereabout. Then they pass into the serum, the agglutinating power of which increases correspondingly. The formation of specific agglutinins in polynuclear leukocytes and in the serum is preceded and accompanied during the first 3 or 4 days after the inoculation of any given microbe by an increase of agglutinins for other microbes. This latter increase, however, is of short duration and stops suddenly, whereas the increase of specific agglutinins persists for a much longer time.

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[1] Jour. of Hyg., II, Nos. 2 and 3, 1902.

[2] Lancet, Feb. 15, 1902.

[3] Centralbl. f. innere Med., XXIII, 145, 1902.

[4] Brit. Med. Jour., April 5, 1902.

The Disappearance of the Addiment from Antimicrobic Serums.—Walker,[1] having shown by animal experimentation that the bacteriolytic addiment of fresh normal serum tends to disappear with considerable rapidity and is no longer present in a serum that has been kept for several days, undertook to examine further the phenomena of its disappearance. For this purpose he immunized a number of rabbits by successive injections of increasing doses of living typhoid cultures, and subsequently bled the animals to collect the serum. The serums thus obtained were examined for bacteriolytic action on Bacillus typhosus, and were compared with fresh serums of 5 normal healthy rabbits. From the results of the investigations it appears that the bacteriolytic power of fresh serum rapidly diminishes both in immune and in normal serums, and ceases to be recognizable within a few days from the time of the bleeding. Hence it appears that as regards experiments in bacteriolysis the age of the serum that supplies the addiment is a factor of the first importance, and that observations on bacteriolytic action with an addiment-containing serum cannot be properly compared unless they are performed at the same time, since the bacteriolytic addiment of a given serum may undergo considerable diminution in a few hours. This fact, it is believed, may throw some light upon the want of harmony in many of the results that have been published, and it affects the value of a number of the recorded observations.

Immunization against Immune Serum.—Walker,[2] with a view to ascertain what results might be obtained from experiments directed to the production of immunity against an immune serum, undertook a series of observations on guinea-pigs with an antityphoid serum of the horse and a variety of Bacillus typhosus. From his studies he concludes that a bacterium may be immunized against its immune serum, and thereby become more virulent and less agglutinable if it be of an agglutinable variety; that the immune serum in which its own bacillus has been grown is rendered less agglutinable and less protective against that bacillus; that agglutinins are true antibodies; that the basis of bacterial virulence and of chemiotactic influence is identical, and constitutes that atom group which causes the production of the immune body; that an animal may be immunized against an immune serum, whereupon it becomes less capable of being protected by that serum, but its susceptibility to the bacterium is not increased; and that the immune body is not identical in different animals for the same bacterium, but exhibits a specialism to the species.

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[1] Lancet, Jan. 4, 1902.

[2] Jour. of Path. and Bact., VIII, 34, 1902.

Bacteriohemagglutinins and Antihemagglutinins.—From a large series of experiments, which are fully described, Karl and Ludwig[1] show that various microorganisms produce, in addition to hemolysins, also bacteriohemagglutinins. Both of these are very unstable and are destroyed at 58° C. The normal serum is not capable of paralyzing hemagglutination, but does paralyze hemolysis; both are paralyzed by specific immune serums. There is no connection at all between the two phenomena. Both processes are separate and may appear independently of each other.

The Influence of Alcoholic Intoxication upon Certain Factors Concerned in the Phenomenon of Hemolysis.—Abbott and Bergey,[2] from an experimental investigation of the question, conclude: (1) The daily administration of alcohol by the mouth to rabbits brings about a reduction in the circulating blood of the hemolytic complement; (2) slight alterations in the normal alkalinity of the blood-serum have no demonstrable influence upon the hemolytic complement of the blood of alcoholized rabbits; (3) the diminished reactivating power of the blood of alcoholized rabbits is not due to the presence of small amounts of alcohol, as such, in the blood; (4) the administration of alcohol to rabbits induces not only a marked reduction in the complement content of their blood, but may cause at the same time a reduction in the specific hemolytic receptor in the blood of rabbits artificially immunized against an alien blood; (5) the diminished complement content of the blood of alcoholized rabbits renders the animal more susceptible to the toxic action of an alien blood.

Serum Reactions of the Blood.—A valuable digest of the literature of the subject is given by Sailer.[3]

The Mode of Action of Various Laking Agents on the Blood-corpuscles.—Stewart,[4] in consequence of much experimental investigation, concludes: (1) Ether dissolves out a considerable amount of material (including cholesterin) from washed formaldehyd-fixed corpuscles. Sapotoxin produces no change in the conductivity of a suspension of such ether-extracted corpuscles. It probably produces the increase of permeability in the unextracted formaldehyd corpuscles by an action on substances soluble in ether. It is to be presumed that it also acts in this way when it lakes unfixed corpuscles. (2) Sapotoxin causes no change in the conductivity of a suspension of washed sublimate-fixed corpuscles. (3) Sublimate-fixed corpuscles can be laked by heating them in water. (4) The addition of ammonia to formaldehyd-fixed corpuscles changes the methemoglobin spectrum into that of oxyhemoglobin (alkaline methemoglobin). The corpuscles can then be laked by the addition of water, slowly in the cold, rapidly on heating. (5) Mercuric chlorid and formaldehyd do not seem to fix precisely the same constituents of the corpuscles. The former fixes the stroma very rapidly. The mercury can be withdrawn from its combination with the stroma by H_2S. On further addition of NH_3 or on adding ammonium sulphid at once, the ghosts may appear. (6) Amyl alcohol in its laking action does not appear to cause the liberation of the electrolytes of the corpuscles. (7) Ether in an amount just sufficient to cause laking does not seem to liberate the electrolytes of the corpuscles, but in larger amount it seems to do so and to break up the ghosts. (8) Some of the agents investigated are active leukolytic agents (especially sodium taurocholate), but there is no direct proportion between their erythrolytic and their leukolytic power. (9) Although some of the laking agents investigated (crude saponin, sapotoxin, sodium taurocholate) increase the permeability of the formaldehyd-hardened corpuscles, and might therefore be supposed to produce their laking action on unhardened corpuscles by increasing their permeability to the dissolved substances of the serum and thus causing water-laking, evidence is brought forward that this is not the primary action of these substances, although it may be a secondary one. For (*a*) laking is produced by all the agents investigated (heat, sapotoxin, sodium taurocholate, foreign serum) in blood to which excess of cane-sugar, which does not penetrate the corpuscles, has been added. Even when blood is saturated with cane-sugar laking occurs. Only in the case of foreign serum is the laking notably delayed and rendered incomplete by the presence of the sugar. Heat-laking is not prevented by sodium chlorid. (*b*) Laking is also produced (by saponin, sodium taurocholate, both added in substance, ethyl alcohol, amyl alcohol, ether, chloroform, and heating to 62° to 64° C.) in sediments of corpuscles freed as far as possible from serum and containing so little intercorpuscular water that even the entrance of the whole of it into the corpuscles could only cause a small amount of water-laking. For example, laking was normal in a sediment containing 7 % to 8 % of serum, and even less.

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[1] Wien. klin. Woch., Jan. 30, 1902.
[2] Univ. of Penna. Med. Bull., xv, 186, 1902.
[3] Univ. of Penna. Med. Bull., xv, 214, 1902.
[4] Jour. of Med. Research, viii, 268, 1902.

On Antibodies against the Bacteriolytic Immune Bodies of Cholera.—Pfeiffer and Friedberger[1] detail the results of some experiments performed with cholera goat-serum preserved since 1895. On two occasions, separated by an interval of 3 weeks, 10 cc. of this serum was injected subcutaneously into 2 rabbits, and 8 days later the blood-serum of the rabbits was tested with reference to its power to arrest the bacteriolytic action of the cholera goat-serum on cholera vibrios in the peritoneal cavity of the guinea-pigs. Mixtures of the serum of the inoculated rabbits, the cholera goat-serum, physiologic salt solution, and highly virulent cholera bacilli were injected into the peritoneal cavities of guinea-pigs. There resulted complete absence of bacteriolytic action of the cholera goat-serum, all of the inoculated animals dying with the usual manifestations of cholera infection. Control tests made with the serum of the rabbits before inoculation with the goat-serum, as well as with the serum of other animals, showed no alteration in the bacteriolytic action of the goat-serum. Subsequently a number of experiments were made with varying doses of "antiserum" and cholera goat-serum, with a view to ascertain the potency of the "antiserum." The series of experiments showed that by injecting rabbits with cholera goat-serum, antiimmune bodies are formed, and that the cholera-immune bodies of the goat and those of the rabbit are specifically different. It is believed also that the amboceptors of normal goat-serum are identical with the amboceptors of specifically immunized goats, and it is suggested that the short duration of passive immunity induced by injecting heteroimmune serum may be explained by the rapid disappearance of injected immune bodies through the formation of "antiserum" bodies, and that the fact that passive immunity induced by isoimmune bodies is of much longer duration may be explained by supposing that the formation of isoantiimmune bodies occurs with greater difficulty.

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[1] Berl. klin. Woch., Jan. 6, 1902.

The Fate of Bacteria in the Organism of Immunized and Non-immunized Animals.—Goldberg[1] performed his experiments as follows: Pure cultures of pathogenic organisms were injected into the blood of animals, and cultures made from time to time to determine the length of time during which the organisms remain in the blood-current, and the degree of their virulence. The animals were finally killed and their organs examined. The following results were obtained: The organisms injected into the blood-current disappeared in from 4 to 8 hours, except anthrax bacilli, which were present in some up to the time of death. Rabbits immunized against Bacillus pyocyaneus became more resistant to injections with anthrax or typhoid bacilli. In the animals immunized against Bacillus pyocyaneus the disappearance of the anthrax bacilli was also inconstant. During the first few hours following the injection the bacilli accumulated principally in the liver, this being more marked in the immunized animals. This retention of the bacteria by the liver is not passive, but depends on active phagocytosis on the part of the endothelial cells and the leukocytes. As infection proceeded the accumulation of the bacteria took place principally in the spleen, where they remained the longest. The organisms retained their virulence within the organs of immunized animals and were capable, under favorable circumstances, of producing local infection (pericarditis, pleuritis, cholecystitis). In the animals immunized against Bacillus pyocyaneus the bacteria accumulated in the spleen, while in those immunized against typhoid they were mostly found in the bone-marrow. The conclusion is drawn that in the former the spleen and in the latter the bone-marrow are the most important defenders of the organism. The agglutinating properties of the blood were not always in proportion to the degree of immunity, and therefore there can be no relation between the agglutinating and bactericidal properties of the blood. The immunity depends principally on the activity of cellular elements of certain parenchymatous organs. Phagocytosis is not the only factor in immunity;

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[1] Bolnitchaia Gazeta Botkina, XII, No. 3; Phila. Med. Jour., Aug. 10, 1901.

the bactericidal properties of the tissues, the antitoxins, and other factors play an important rôle.

Coffee and Tea as Precipitants for Poisons.—Sollmann[1] gives the following as the results of his investigations: (1) Precipitation of alkaloids: Atropin, coniin, morphin, and pyridin are not precipitated even in fairly strong solution by coffee. Tea precipitates them from strong but not from weak solutions. Aconitin, brucin, cocain, lobelin, nicotin, and pilocarpin in weak solution are only sparingly precipitated by tea; coffee does not affect them even when they are in concentrated solutions. Apomorphin, the cinchona alkaloids, hydrastin, strychnin, and veratrin in dilute solutions are precipitated efficiently by either coffee or tea, the latter being generally more efficient, except perhaps for veratrin and quinin. The precipitation is incomplete with all alkaloids except apomorphin. However, the quantity of unprecipitated alkaloid is quite small in those which are said to precipitate from "dilute solution," since most of the alkaloid is removed from 1 : 2000 solutions. The precipitates are somewhat soluble in dilute HCl, very readily soluble in dilute alcohol. The administration of the latter therefore must be avoided if these beverages (or tannin) are used in chemic antidotes in alkaloidal poisoning. Since the precipitates are not quite insoluble in water, as little liquid as possible should be given. The quantities of the decoctions should not be less than 3 cc. of a well-boiled 10 % decoction for each milligram of the alkaloid. (2) Metallic salts: Tea is also the more efficient precipitant of metals, but the difference is not nearly so striking as with alkaloids. Both beverages are inefficient against arsenious acid or tartar emetic. They precipitate to a large extent, but not quite completely, the salts of cobalt, copper, nickel, uranium, and zinc,. and would be useful antidotes against the toxic members of this list. They precipitate practically completely the salts of aluminium, lead, and silver. Mercury is partly precipitated by tea, but not by coffee, so that the former would be an antidote, the latter not. (3) Proteids: These differentiate very sharply between the two tannins: whereas tea produces large precipitates, coffee leaves them unaffected, or renders them slightly turbid at most. This serves to explain the less astringent taste of coffee and its less deleterious effect upon digestion. The reactions of tea bear a very close resemblance to those of gallotannic and quercitannic acid. The precipitant effects of caffeotannic acid are weaker, but occur along the same lines. The greatest differences are seen in their action on proteids and on certain alkaloids, whereas other alkaloids and most metallic salts are precipitated almost equally well by both. An exception is formed by mercuric chlorid, which is partly precipitated by tea, not at all by coffee.

The Poison of Russell's Viper (Daboia Russellii).—Lamb and Hanna,[2] having studied the action of Russell's viper, conclude: (1) Daboia venom owes its toxic property principally, if not entirely, to its action on the blood; it causes rapid death from extensive intravascular clotting, while in the chronic cases a marked deficiency of the blood coagulability is observed. To this deficiency are probably due the hemorrhages, both actual and serous, which are such prominent symptoms in such cases. (2) Heating a weak solution of daboia venom—*i. e.*, a 0.1 % solution—for half an hour at 73° C. completely destroys the toxicity. When the solution is more concentrated—*i. e.*, 1 %—the power of causing intravascular thrombosis is not destroyed but only lessened. (3) Daboia venom contains no elements possessing a similar action to the albumose of cobra venom, which is the chief constituent of the latter, and which acts directly on the central nervous system. On the other hand, cobra venom contains no substance which can cause intravascular thrombosis. (4) Calmette's serum has no power to neutralize the toxin of daboia venom.

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[1] Jour. of Med. Research, VII, 43, 1902.
[2] Jour. of Path. and Bact., VIII, 1, 1902.

Snake Venom in Relation to Hemolysis, Bacteriolysis, and Toxicity.—Flexner and Noguchi[1] have studied the action of dried venom from the standpoint of agglutination and the dissolution of both its red and white corpuscles. Their conclusions from these experiments are as follows: (1) Venom contains principles which are agglutinating and dissolving for white blood-corpuscles; (2) the agglutinating principle may be identical for both white and red cells; (3) the dissolving principle for leukocytes is distinct from that for red cells; (4) in order that solution of venomized leukocytes shall occur a complement-containing fluid is required; (5) the several varieties of white cells of the rabbit's blood show different susceptibilities to the action of venom, the lymphocytes showing the least. Their experiments also showed that the brain contained more venom-neutralizing substance than any other organ or tissue in the body. Animals injected with twice the minimal lethal dose in sterile brain emulsion lived 19 hours, in blood-solution 3 hours and 50 minutes, etc. The neurotoxin and hemolytic principles are physiologically distinct. Although the chief toxic constituent unites with the nerve-cells, still multiple minimal lethal doses, from which the neurotoxic principle has been removed, may contain sufficient hemolysin to bring about fatal intoxication. In determining the effect of venom upon the bactericidal properties of the blood they have found: (1) that all venoms, when used in suitable quantities, destroy the bactericidal properties of many normal blood-serums; (2) this destruction consists in the fixation of the serum complements by the venoms; (3) the venoms have no action upon the intermediary bodies of serum; (4) if the venom is incapable of uniting with the serum complements, then the original bactericidal properties remain unaffected by the presence of the venom. When Calmette's antivenene was used to the extent of neutralization of the venom, the erythrolytic, leukolytic, and antibacteriolytic actions of the venom were removed.

Antivenene.—McFarland,[2] in consequence of considerable experimental investigation and a study of the literature, concludes: (1) It is not difficult to produce immunity to the nervous poison contained in serpent's venom, as shown by Wolfenden, Phisalix, Bertrand, and Calmette; (2) this immunity, when carried to a high degree, is associated with a marked antitoxic power of the blood; (3) it is very difficult, if not impossible, to produce immunity to the irritative poison of the venom; (4) antivenene does not protect very powerfully against the irritative poison; (5) antivenene protects against the nervous poisons of various venoms; (6) immunity to the unmodified venom is better secured by intravenous than by subcutaneous injection; (7) Calmette's method of immunization with the modified venom is greatly preferable to the method of immunization to the unmodified venom as tried in these experiments, because of the modicum of danger and suffering to the horses used; (8) Calmette's antivenene is more useful for the treatment of the bites of cobras and colubrine serpents than for those of vipers, etc.; (9) as the antivenene protects against the chief death-dealing element in the venoms, it is of great use in the treatment of all serious bites, and should be used whenever possible; (10) the neutralization of the nervous poison of the venom by antivenene will enable the individual to devote all his vitality toward overcoming the local injury done by the irritative globulin in the venom; (11) all individuals—certainly not all horses—are not equal in their resisting power against venom; 2 of my 3 horses succumbed quickly to the venom injections; (12) the antivenene-producing power varies in different horses; (13) the quantity of antivenene in the blood varies from time to time, according to circumstances.

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[1] Jour. of Exper. Med., Mar. 17, 1902; Univ. of Penna. Med. Bull., Feb., 1902.

[2] Jour. Am. Med. Assoc., xxxvii, 1597, 1901.

Food Intoxication and Infection of Unique Origin.—Ohlmacher[1] reports the details of a food infection involving 228 inmates of an institution. The infection was traced to oatmeal that had become contaminated with plaster-dust from the ceiling of a room being repaired and in which the oatmeal was allowed to cool after being cooked. Bacteriologic examination of the ceiling revealed Bacillus coli communis and Proteus vulgaris, both of which were found to be pathogenic and both of which are credited with causing the infection. Toxic substances were isolated from some oatmeal artificially contaminated with the plaster-dust.

Classification of Intoxications.—Adami[2] proposes the following classification of intoxications from a pathologic standpoint: (1) Exogenous, due to the actions of poisons entering the system from without. (*i*) Exotic or introduced: due to the action of substances foreign to the organism, which gain entrance through the skin, the digestive, the respiratory, or the genitourinary tract. (*ii*) Indigenous or excretory: (*a*) Indirect autointoxication, due to the absorption of retained excreta; (*b*) disintegrative, due to the absorption of decomposition and fermentation products developed in the external secretions through the action of those secretions. (2) Endogenous. (*i*) Direct autointoxication: (*a*) Internal secretory, due to the action of excessive or unneutralized or modified discharges from the cells of one or other tissue acting directly upon the other tissues of the body without previous discharge from the system: (*b*) disintegrative, due to the action of the products of decomposition and necrosis of one or other tissues acting in a similar manner. (*ii*) Parasitic: (*a*) Microparasitic, the infections; (*b*) macroparasitic.

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[1] Jour. of Med. Research, VII, 411, 1902.

[2] Amer. Med., II, 131, 1901.

Observations Concerning the Production of Plasmotropic Poisons in the Organism.—Grawitz[1] differentiates between poisons having "plasmotropic" and "plasmolytic" effects on blood-cells. Under the former heading he classes those which do not destroy red corpuscles in the circulatory stream, but produce their destruction in increased quantities in liver, spleen, and bone-marrow without causing direct lysis, associated with extrusion of hemoglobin into the plasma. Under plasmolytic poisons he refers to those which dissolve the red corpuscles within the blood, liberating the hemoglobin and producing hemoglobinemia, hemoglobinuria, etc. The type of the former is lead; it produces a granular degeneration of the erythrocytes, but never a plasmolysis in the circulation; in even the worst cases it only destroys large quantities within the organs. He found the same basic degeneration in cases of hemorrhage from internal organs, as gastric ulcer, carcinoma, cirrhosis of the liver, etc. It was not found in the cases in which the hemorrhage was external. The thought arose that this might be due to a formation of plasmotropic poisons while the blood was passing through the intestines. He fed patients with products of hemoglobin, thus producing the same degeneration. He concludes from this that blood-derivatives when absorbed into the blood-stream have plasmotropic effects.

The Influence of the Spleen on Natural or Acquired Hemolytic Properties of Blood-serum.—Levin[2] believes that the spleen is undoubtedly not indispensable for the acquired or the natural hemolytic properties of the blood-serum, nor, so far as his experiments go, does it appear to elaborate those substances upon which either the normal or the artificial hemolytic properties of the blood-serum depend.

TUMORS.

The Etiology of Carcinoma.—Although the study of the etiology of carcinoma has claimed the attention of many investigators during the past year, the general state of the question remains much as it was a year ago.[3] The most important contribution to the literature of the subject has been the **second annual report of the Cancer Commission of the Surgical Department of the Harvard Medical School.**[4] Tyzzer, as a result of a study of **coccidium infection of the rabbit's liver,** concludes: (1) Associated with certain lesions in the liver of the rabbit are found parasites, of varying form and character. (2) The various forms represent the life-cycle of a definite species of sporozoon, Coccidium oviforme. The necessary stages of the life-cycle are traceable. (3) In only one stage does the parasite resemble the cell-inclusion of cancer. Even this stage presents a definite and constant morphology. (4) The immediate effect of the parasite upon the host is to produce degeneration and destruction of the epithelial cells of the bile-ducts. Secondary to this, the effects of irritation are seen in the proliferation of the connective tissue and epithelium. The most remote changes are those of cirrhosis. (5) Repair is effected through walling-off of the process by connective tissue, by the destruction of the remaining parasites, and finally by cicatrization. There is no well-founded analogy between the cell-inclusions of cancer and any one of the stages in the life-history of Coccidium oviforme. The lesion is of the nature of a chronic inflammatory process. The tissues react to the irritation which the parasites cause by their presence in the bile-ducts. With the removal of the irritation, repair takes place. Thus the whole is to be regarded as a physiologic process, checking the inroads of the parasite. It has little in common with new-growth. No metastases are formed, nor is it possible for them to be formed, as is evident from the nature of the lesion. The process is self-limited and repair follows the destruction of the parasites. White and Robey, as a result of a study of **molluscum contagiosum,** conclude that no one has demonstrated any parasitic body in the growth, and that the change is not a colloid or hyaline degeneration, but rather an extraordinary metamorphosis of the rete cells into keratin. Richardson, undertaking an additional series of **culture experiments with malignant tumors,** confirmed his previous observations, being unable to cultivate from the tissues or fluids of malignant new-growths anything which could be regarded as a specific infecting microorganism. Weis gives the result of his investigations of **4 pathogenic torulas,** more commonly known as **blastomycetes,** and reaches the conclusion that they cannot be considered to have anything to do with the etiology of carcinoma. Nichols gives a critical study of the literature of **the relation of blastomycetes to cancer** and the results of considerable personal investigation, concluding: (1) Certain blastomycetes can live and multiply in human and animal tissues, produce local lesions, and metastases in the internal organs; that is, they are pathogenic. (2) The lesions produced in animals by spontaneous infection with blastomycetes are acute inflammation, abscesses, or nodules of peculiar granulation tissue, and are not in the least analogous to cancers. (3) The lesions produced in human beings in cases of spontaneous infection with blastomycetes are acute inflammation (abscesses or ulcers) or proliferation of endothelium and connective tissue. At times a proliferation of the epidermis does occur, but is not due to the action of the blastomycetes, but is secondary to the chronic inflammation of the underlying corium. This proliferation of epidermis is not analogous to the proliferation of epithelium seen in cancers, since no epithelial metastases occur. (4) Blastomycosis in human tissues is very rare. (5) The lesions produced in animals by experimental inoculation with blastomycetes are, with the exception of Sanfelice's "successful" cases, inflammations or nodules of peculiar granulation tissue. Sanfelice's cases are not conclusive in themselves, are in direct opposition to the results obtained by all other observers, and, even if true, are logically explained as coincidences, and not as results. (6) Blastomycetes as a rule cause marked proliferation of tissue, and little infiltration with leukocytes; that is, their toxic powers are small. (7) Blastomycetes primarily extend along lymphatic clefts and vessels. (8) Rarely in human beings, more often in spontaneously infected animals, and often in experimental animals, blastomycetes may be taken into the blood-vessels, disseminated throughout the body, and produce a general infection and metastases. (9) The secondary nodules have the same general character, that is, a formation of granulation tissue, as the original nodules. (10) The morphology of the so-called "cancer bodies" is not identical with that of the blastomycetes. (11) Blastomycetes are not constantly present in human malignant tumors and cancers. (12) Even if blastomycetes do occur in human cancers they are not present in such numbers and in such a relation to the anatomic lesion as to justify the belief that they are the cause of the disease. All of which facts lead to the conclusion that there is no evidence that blastomycetes have anything to do with the production of human cancers. Greenough gives the results of a further study of **cell-inclusions in cancer and in noncancerous tissue,** stating: (1) Cell-inclusions of a constant type are found in practically all cases of cancer of the mammary gland; (2) they are also found in noncancerous tissue of the mammary gland; (3) they are not found in epithelioma or in sarcoma; (4) their appearance, staining reactions, and situation in the cell are such as to justify the hypothesis that they are the result of the secretory activity of the epithelial cell; (5) there is no cause for attributing a parasitic origin to their appearance. Concluding, Nichols states that it has been claimed by the adherents of the theory of the parasitic origin of cancer that: (1) A proliferation of epithelial cells analogous to the lesions seen in cancerous tumors can be produced by certain well-known protozoa (nodules caused by the Coccidium oviforme); (2) certain skin lesions characterized by epithelial cell proliferation are due to the action of a so-called protozoa (molluscum contagiosum); (3) blastomycetes are constantly present in human cancers and are the cause of the lesion; (4) by experimental inoculation of animals with "blastomycetes" true epithelial or cancerous nodules can be produced; and (5) the well-known endocellular bodies seen in the protoplasm of cancer cells have a definite morphology, are "parasites," and the cause of cancer. As a result of the work pursued by the Cancer Commission, it is concluded: (1) The lesion produced by Coccidium oviforme is essentially a process of chronic inflammation and is not analogous to the lesion seen in cancer; (2) the lesion in molluscum contagiosum is characterized by certain changes in the epidermis, is not due to the action of a protozoon, and is not analogous to cancer; (3) the so-called "blastomycetes" ("saccharomycetes") of Sanfelice and Plimmer are torulæ; (4) the lesions produced by these "blastomycetes" (torulas) are essentially nodules of peculiar granulation tissue, are not cancerous, nor in any sense true "tumors"; (5) blastomycetes are not constantly present in human cancers; and (6) the peculiar bodies seen in the protoplasm of cancer cells are not parasites, nor the cause of the lesions, but probably are in part at least atypical stages of the process of secretion by glandular epithelium. **The analogies existing between Plimmer's bodies and certain structures normally found in the cytoplasm**—centrosomes, attraction spheres, ectospheres, entospheres, etc.—are pointed out by LeCount.[1] From his study he believes that we are warranted in requiring enthusiastic advocates of the parasitic theory of carcinoma, who use Plimmer's bodies as a support for their views, to exclude or to account for the centrosomes and the enveloping variously-named formations of the archoplasm in carcinoma cells.

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[1] Deut. med. Woch., Dec. 26, 1901.
[2] Jour. of Med. Research, VIII, 116, 1902.
[3] See the YEAR-BOOK, vol. I, p. 385.
[4] Jour. of Med. Research, VII, 235–382, 1902.

A Possible Predisposing Cause of Carcinoma.—Mason[2] adduces a number of statistics and observations that he believes indicate that, whatever may be the ultimate cause, carcinoma will be found to be due to some germ whose habitat is a sewage-contaminated subsoil, and that its entrance into the body is through one of the apertures lined with mucous membrane.

Etiology of Carcinoma and Autoimplantation of Carcinoma Cells into the Walls of the Stomach and into the Lungs.—Fütterer's[3] conception of the origin and development of carcinoma is as follows: Normal epithelial cells, which are exposed to chronic disorders of circulation, undergo various changes in form and function. Their method and time of multiplication are also changed, and this leads to abnormal grouping of cells. Proliferating epithelial elements under the influence of a mechanical trace are pressed into the deeper tissues accompanied by sprouting blood-vessels and surrounded by hyperemic and edematous areas and more or less cellular infiltration. Here they form the body of the primary tumor, and from here some of their cells are carried to distant parts mainly by the lymphatics. Many cell embolisms are lost, while others lead to development of metastases through a proliferation of their own cells, which become inclosed in the meshes of framework formed by the proliferating connective tissue and the sprouts of blood-vessels of the organ invaded. The cells of the primary growth may develop noticeable physiologic activity, manifesting itself also in the metastases of physiologic function, which may not be recognizable in the primary growth, but again is plainly discernible in the metastasis. The primary growth may be physiologically active; not so the first metastasis, but the next metastasis may exhibit clear evidence of physiologic activity. By physiologic activity is here meant demonstrable physiologic activity, since it is probably always present in carcinoma cells. Observations show that carcinoma cells may retain the physiologic function of the cells from which they are derived. He cites 5 instances of physiologic activity on the part of metastases removed some distance from the primary seat. Concerning autoimplantation of detached carcinoma cells into distant parts of the human body, he has observed 2 cases. The first is a flat-celled carcinoma of the esophagus, with ulcerating surface, from which cells have become detached, and on entering the stomach have been pressed into the mucous membrane of that organ, giving rise to an implantation carcinoma. Continuity, contiguity, and metastasis are clearly excluded by macroscopic and microscopic findings. But 6 clear cases of implantation from one part of the digestive system to another are recorded in literature, this making the seventh one. His second case concerned a carcinoma of the thyroid gland, which broke into the trachea and from which particles had become implanted into lung, metastasis occurring from these implantations to the pleura. But two such observations are recorded.

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[1] Jour. of Med. Research, VII, 383, 1902.

[2] Brit. Med. Jour., Jan. 18, 1902.

[3] Medicine, 1902, VIII, 177.

Excess of Salt in the Diet a Probable Factor in the Causation of Cancer.—Braithwaite[1] thus explains his theory of the causation of cancer: (1) That an excess of salt in the diet is one of four factors which originate the disease. This is the essential factor, but it is inoperative without at least one, and probably two, of the others. Excess of salt may arise from individual taste, or from much salt meat, or from too much ordinary meat, which of course involves much salt. The other factors are these: (2) An overnourished condition of body from more food, and especially more meat, than is required. This condition is rarely met with among out-of-door manual workers. (3) An impure condition of body owing to nonuse and nonoxidation of the food which has been taken. The amount may have been moderate or even small. The cells of the body in this condition are loaded with effete material. It obtains in old age; in persons who lead indolent, easy, and indoor lives; and locally in organs the active functions of which have ceased. (4) A fourth factor is some local irritant or stimulant, such as friction from the stem of a pipe or irritation from some microorganism of which no one is actually specific, or ovarian stimulation in the case of the breast. Of these, the first must always be present, and probably in some form the fourth, and also in all either the second or the third, but not both of them. These factors being in existence, the disease may be started in perhaps one epithelial cell or in a mass of cells which grow individually larger and change the nature of their protoplasm, for a cancer cell will not stain with congo-red, while an epithelial cell takes the stain deeply. The cell becomes a different being with often more than one nucleus. It is itself the parasite, living and growing at the expense of the tissues around it, and contributing nothing to the common good.

The Pathology of Catabolism in Relation to the Etiology and Pathology of Cancer and Allied States.—Wakefield,[2] pointing out that an expression of normal cellular activity consists in the production of sarcolactic acid which becomes almost immediately neutralized by the alkaline intercellular juices, and that health is represented by a balance between metabolic constructive and destructive processes, says that divers pathologic states may result from excessive formation of sarcolactic or other acids or from defective neutralization or oxidation. The greatest morbid influence is attributed to defective neutralization (subcatabolism, or catabolic stasis), a condition manifested by inactivity, devitalization, and premature senility of the cell, and due to suboxygenation, hyperacidity, absence of sun, light, and heat, and excessive strain and fatigue of the cerebral, nervous, muscular, and fibrous tissues. This catabolic stasis is believed to manifest itself locally in persistence of the cells in a superannuated form in hypertrophies and hyperplasias of the fibrous and cellular structures (local suppurations, tumor formations, etc.), while the general or systemic manifestations consist in different degrees of cachexia, pigmentation of the glands and the skin, petechias and angiomas, catarrhal conditions of the mucous membranes, and a predisposition to general suppuration on slight provocation. It is believed that cancers and allied states are not supernourished processes, as the apparent vascularity would suggest, but are really deficient in exchange of both blood and lymph, nor are malignant growths necessarily of bacterial origin. When bacteria become the causative factors, they do so by acting, either as irritants causing sarcolactic acidity, or by their acid excretions, acting directly and in conjunction with sarcolactic acidity. The sum total of all bacterial activity is the production of subcatabolism, the direct causative factor of cancers. Finally, it is said that cancer is both a local and a constitutional disease. The local cause determines the site, while general and systemic are the hyperacidities and suboxidations that lower catabolism sufficiently to inhibit the catabolic dissolution and oxidation of locally produced stases. The arrest of dissolution of the maternal nuclei after division and following an acid-produced retardation of catabolism is said to give rise to changes, by virtue of which they present appearances which have been mistaken for protozoa, blastomycetes, and other unicellular organisms.

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[1] Lancet, II, 1578, 1901.

[2] Amer. Med., IV, 809, 851, 1902.

The General Pathology of Tumors.—C. P. White[1] thus summarizes his opinions with regard to the pathology of tumor formation: (1) Tumors are to be classified on a histologic basis. The best mode of effecting this is to make use of the threefold basis of cells, tissues, and organs. (2) The rudiment from which a tumor springs may consist (*a*) of the structures normally present at the point of origin; (*b*) of an embryonic collection of cells such as is described by Cohnheim; or (*c*) of tissues of new formation, the result either of an inflammatory condition or of previous tumor formation. (3) Extrinsic factors play a part in tumor causation, but are not the determining factors—that is, the occurrence or nonoccurrence of a tumor does not depend on extrinsic factors. In particular, the parasitic theory is shown not to stand a critical investigation. (4) The determining factor in tumor causation is to be found in the intrinsic factors. (5) This determining factor consists in the existence of a condition of unstable equilibrium between the intercellular forces, so that proliferation, once started, is progressive and is not limited by the resistance of the surrounding tissues. (6) The causes of this instability are many and various and may be either intrinsic or extrinsic. (7) Proliferation having started, the cells acquire the habit of growth—that is, the power of independent proliferation which enables them to proliferate in parts of the body in which the condition of equilibrium is stable. (8) Tumors grow by proliferation of their own cells. (9) Tumors do not invariably continue to increase without limit. Under certain circumstances they may cease to grow and may diminish in size or may even disappear completely. (10) Tumor formation is not apt to be regarded as an isolated process, but is to be considered as one of a group of progressive processes with which it is closely allied. Still less must one form of tumor, such as carcinoma, be considered apart from the others. White proposes a new **classification of tumors,** as does also Adami.[1]

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[1] Lancet, I, 494, 1902.

Multiple Primary Malignant Tumors; a Primary Sarcocarcinoma in the Thyroid of a Dog with Mixed Sarcomatous and Carcinomatous Metastases.—Wells[2] points out that combinations of carcinoma and sarcoma in the same individual are of two classes: (1) That in which each form of tumor occurs as a separate growth, either distinct from each other or close together, but having no structural relation; and (2) that in which the neoplasm presents the two distinct types of malignant growth in combination in a single primary tumor—carcinosarcoma or sarcocarcinoma. Reviewing, then, the literature and reporting a personal observation, he concludes: (1) Carcinoma and sarcoma may occur simultaneously in the body of an individual as separate growths. This combination is no more frequent than is readily explained by assuming it to be a mere coincidence, and there is no evidence that the presence of carcinoma in any way predisposes the individual to the development of sarcoma, or conversely. (2) Sarcocarcinoma, a tumor composed of malignant growth of both types, is even more rare. But 3 authentic cases can be found, and each of these originated in the thyroid. Metastases were studied in only one, and here not only were some of the secondary growths of mixed type, but pure sarcomatous and pure carcinomatous secondary growths were also found. (3) These facts may be of interest in considering the etiology and biology of malignant neoplasms, but they do not offer a direct explanation of any of the existing problems. Warthin[3] reports a case of **multiple primary neoplasms in one individual**—spindle-cell sarcoma of the forearm, adenocarcinoma of the pylorus, and myomas of the wall of the stomach. McCrae[4] reports a case of **round-cell sarcoma of the stomach** with secondary manifestations in an already **adenomatous thyroid.**

Primary Sarcoma of the Thyroid Gland.—Lartigau,[5] reporting a case of angiosarcoma developing in a goiter and resulting in death from asphyxiation from pressure on the trachea, reviews the literature of primary sarcoma of the thyroid (51 cases). Though primary sarcoma of the thyroid is rare, it is believed to be more common than statistics show. It is less frequent than primary carcinoma of the thyroid—1 to 3 or 4. It is commonly associated with goiter. Those cases developing in persons between 40 and 60 years of age show a higher percentage of previous goiter than younger persons. Goiter associated with sarcoma is more common in women than in men. Sarcoma of the thyroid occurs oftener in late than in early life—the greatest age-frequency being between 40 and 60 years. Sex is probably unimportant. The primary tumor most frequently originates in the right lobe, and more frequently thus in men than in women. The disease usually runs an acute course, and commonly leads to involvement of the trachea or larynx by pressure or invasion. Metastasis occurs through the blood-vessels or lymph-channels or both. Round-cell, spindle-cell, or mixed-cell sarcomas are most common, though angiosarcoma is not rare.

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[1] Jour. of Path. and Bact., VIII, 1902.
[2] Jour. of Path. and Bact., VII, 357, 1901.
[3] Phila. Med. Jour., IX, 701, 1901.
[4] N. Y. Med. Jour., April 12, 1902.
[5] Am. Jour. Med. Sci., CXXIII, 156, 1901.

Tubular Perivascular Sarcoma.—Low and Lund[1] give a thorough review of the different views entertained with regard to the origin, structure, and metastases of tubular perivascular sarcoma, and report in detail several illustrative cases.

The Myeloid Tumors of Tendon-sheaths.—Bellamy[2] reviews the literature of the subject and reports a personal observation—a tumor consisting of 2 kinds of cells—small cells constituting the main mass of the growth and large multinucleated or giant-cells; adult connective tissue; vessels whose endothelium was in a condition of active proliferation; and showing an advanced stage of fatty degeneration. It is believed that the tumor resulted from proliferation of the endothelium of the blood-vessels, therefore an endothelioma; and in view of the large number of giant-cells the term "myeloid endothelioma" is suggested. It is said also that giant-cells such, for instance, as occur in this tumor, since they result from causes incidental to the pathologic anatomy of the tissue in which they occur, cannot be looked upon as being in the same category as the cells normally present in the bone-marrow.

A fibrocystic tumor of the breast in which the majority of the cysts were lined with **stratified epidermis-like cells** is reported by Griffiths.[3]

A peculiar cyst on the anterior chest-wall between the fibers of the pectoral muscle is reported by Condon.[4] The likelihood of the cyst being a bursal cyst (such as has been described by Chassaignac) or a cystic degeneration of a subpleural lymph-node is excluded, and it is concluded that in the beginning the cyst was probably a portion of parietal pleura which at the time of an injury to the chest-wall was cut off from its neighboring tissue and displaced from its natural position.

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[1] Jour. of Med. Research, VII, 83, 1902.
[2] Jour. of Path. and Bact., VII, 465, 1902.
[3] Jour. of Path. and Bact., VII, 447, 1901.
[4] Jour. of Path. and Bact., VII, 368, 1901.

A case of enchondroma-like formations in the femur following osteomyelitis is reported by Block.[1]

Verrucose Nevus Associated with Certain Anomalies of Pigment.—Taylor[2] records a case of verrucose nevus, occurring in a boy aged 19 years, and interesting particularly on account of the manner in which the lesions were rigorously confined to the right side of the body, their distribution in the track of certain cutaneous nerves, the alternation of deeply pigmented papillomas with merely the presence of pigment in the skin, and the merging of this pigmented condition with decolorized patches. The lesions occurred on the right side of the chest, to the right of the umbilicus, on the right side of the scrotum and penis, and on the right side of the nose, forehead, and chin.

Lymphomatous Tumors of the Dog's Spleen.—Williams and Busch[3] describe the results of their study of lymphomatous nodules found in 17 spleens encountered at the necropsies performed on 720 apparently healthy dogs. The number of tumors seen in a single spleen varied from 1 to 7; they were roughly spherical in shape, and from 0.4 cm. to 4 cm. in diameter. Macroscopically they consisted of small, soft, white, translucent masses resembling malpighian bodies, embedded in a soft, dark red substance, looking like spleen-pulp or blood-clot. The nodules differed from the normal spleen in the absence of trabeculas except at the edges, in the large size of the lymphoid masses as compared with the malpighian bodies of the normal spleen, and in the absence of a central artery in the lymphoid masses as a rule, though not invariably. The superficial resemblance of these tumors to hemorrhages into the dog's spleen (hæmorrhagica intrapulposa subcapsularis, Kitt) is pointed out, and the means of differentiation suggested. A traumatic origin of the tumors being among the possibilities, experimental lacerations were undertaken, with negative results. The tumors observed are believed to correspond with the lymphoma, or circumscribed tumors of lymphoid tissue, of Kitt.

Dermoid Cysts and Teratomas of the Anterior Mediastinum.—Christian,[4] from a study of a case and a review of the literature (40 cases), states that dermoid cysts and teratomas of the anterior mediastinum occur infrequently, generally give evidence of their presence during early life, and are of relatively long duration. Their most frequent position is immediately behind the upper portion of the sternum, whence they may grow into the lung or downward between the heart and lung. They may be classified as follows: (*a*) Tumors of ectodermal origin with the addition of some tissues from the mesoderm—dermoid cysts; (*b*) tumors derived from all 3 layers—teratomas; and (*c*) dermoid cysts or teratomas which in some part of their structure show evident malignancy. Most of the reported cases belong to the first-mentioned class. The coughing up of hair is pathognomonic of their presence. Cure is possible only through surgical procedures. The genesis of the growths is to be referred to fetal life. Most of them are branchiogenic in origin. Some may result from germ layers misplaced at the time of closure of the anterior chest-wall.

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[1] Jour. of Path. and Bact., VII, 227, 1902.

[2] Lancet, Jan. 11, 1902.

[3] Jour. of Med. Research, VII, 408, 1902.

[4] Jour. of Med. Research, VII, 54, 1902.

A tumor-like lesion in the lung of a horse caused by a blastomyces (torula), believed to be the only case so far recorded, is reported by Frothingham.[1] The lesion consisted of a mass of tissue, much resembling a myxosarcoma, about the size of a human head, and containing large and small areas of necrosis. Microscopically it consisted of delicate trabeculas of connective tissue which formed a network in the meshes of which was a varying number of cells, some probably endothelial, others connective tissue, and a great number of blastomycetes. The organism, believed to be a torula, was isolated in pure culture; and both the culture and material from the original lesions, when injected into rabbits and guinea-pigs, produced similar lesions, and if time permitted metastases. Whether or not this torula is identical with Klein's, Plimmer's, or one of Sanfelice's is not yet determined. It produces, however, a purely inflammatory reaction, and not a proliferation of epithelium.

Transplantation of Tumors.—Loeb[2] gives the details of his continued investigations of the transplantation of tumors, especially of the successful transplantations of sarcomas found in white rats into animals of the same species. It is believed that many peripheral sarcoma cells remain alive in the first few days after transplantation, and that they mix with the growing connective tissue of the neighborhood, and that these peripheral sarcoma cells give rise to the tumors after transplantation. It is said also that the growing sarcoma has the power to penetrate into the central necrotic part of the transplanted piece, which becomes liquefied, probably under the influence of proteolytic ferments. In a second series of investigations all attempts to produce tumors by transplanting pieces or by injecting tumor fluid into white mice, guinea-pigs, and rabbits were without result, and in a new series of investigations injection of cystic fluid or of fluid obtained by mincing the tumor with normal salt solution did not produce sarcomas. A series of experiments was undertaken to determine how long pieces of tumors could be kept on ice and yet develop sarcomas after transplantation, with the result that it was found that pieces kept on ice for 1, 2, and 5 days developed in each case several tumors, whence it is concluded that the tumor-producing agency, as well as the sarcoma cells, are not destroyed or even markedly attenuated by being kept on ice for 5 days. The result of transplanting an adenoma of the mammary gland differed according to the animal experimented with. Pieces transplanted into the same animal remained alive *in toto* and underwent afterward the same cycle of changes as the part of the original tumor that was allowed to remain. Pieces transplanted into other rats became almost entirely necrotic. In one of these pieces, however, some peripheral parts of the glands remained alive for a few weeks and did not even grow by mitotic division. In the original piece, and in the piece transplanted into the same rat, pregnancy induced a large increase in the size of the tumor, especially in the glands, and probably also some mitoses. Mast-cells present in the tumor were successfully transplanted. Herzog[1] makes a preliminary report upon a number of successful **transplantations of tumors** in rats.

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[1] Jour. of Med. Research, VIII, 31, 1902.

[2] Jour. of Med. Research, VIII, 44, 1902.

The Growth of Epithelium in Agar and Blood-serum in the Living Body.—Loeb,[2] detailing the results of his continued investigations of the growth of epithelium, states that by transplanting epithelium into agar or blood-serum in the living animal changes can be observed resembling changes taking place in carcinomatous epithelium. Of the structural peculiarities thus produced, some are caused by degenerative processes in the epithelial cells, others by inclusion of foreign particles in the cells, still others by foreign bodies determining the arrangement of the epithelial cells (formation of epithelial pearls). These changes will aid us in determining what changes in carcinoma are produced by degeneration of carcinoma cells and what changes may be produced by the presence of foreign bodies. The inclusion of blood-serum in epithelial cells can be observed as early as 5 days after operation, and can still be seen 12 days after operation. Pigmented epithelial cells transplanted into blood-serum show in the first 12 days the same changes in pigmentation that were found to take place in epithelium when it is regenerated in the usual way, together with connective tissue. The more pronounced degenerative changes in epithelium transplanted into blood-serum begin to take place about the eighth day after transplantation. As a result of his experiments Loeb believes that the changes found in carcinoma (a part of which may also be found in blastomycetic dermatitis) are not, as far as they are referred to in the paper, caused by a specific influence (for instance, blastomycetes), but are determined by the character of the epithelial cells, under the influence of chemically indifferent foreign substances.

Carcinoma and Tuberculosis.—Moak[3] reports 5 cases of carcinoma and tuberculosis in the same organ or tissue, and gives a valuable discussion of the subject.

Oidiomycosis (Blastomycosis) of the Skin and its Fungi.—Ricketts[4] details the results of a most exhaustive study of oidiomycosis, comprising: The history and pathogenic properties of oidia (in which he discusses blastomycetes and saccharomycetes in human and animal lesions other than blastomycetic dermatitis, experimental blastomycelial and similar infections in animals, the blastomycetic theory of tumors, and Sporothrix schenckii); a summary of so-called blastomycetic infections in man (discussing Saccharomycosis hominis—Busse, Saccharomycosis humaine—Curtis, blastomycetic dermatitis—Gilchrist, protozoic infection of Wernicke and others); the clinical, mycologic, and histologic study of 12 cases of oidiomycosis of the skin; a critical analysis of blastomycetic infection of the skin; and an extensive bibliography. He points out that it has been generally considered that thrush is the only example of a disease caused by an oidium, the process being limited largely to the mucous membranes of the mouth, pharynx, and esophagus. Observers have shown, however, that the parasite of thrush may penetrate to the submucosa of the pharynx, esophagus, and stomach, and even produce thromboses with consequent ulceration. Of greater importance are the observations of abscesses in the brain, liver, and kidney, and of abscesses and areas of consolidation in the lungs, the conditions being associated with the presence of organisms of the oidium type. In most of such cases there was a concomitant mucosal thrush, and, so far as stated, there was no mixed infection in the abscesses. In addition, organisms not differing essentially from Oidium albicans have been cultivated from cases of tonsillitis, thrush, and infections in other parts of the body, and have been termed saccharomycetes or blastomycetes by their observers. The natural pathogenicity of organisms of this type extends to certain animals, notably horses, which in Japan, Italy, Russia, and some other countries suffer epidemically from a stubborn burrowing infection of the skin, superficial lymphatics, and upper respiratory tract. Recently especial interest has been taken in such infection of the skin in man. Permanent cultures of the organism were obtained from 7 out of 9 cases examined bacteriologically. It is stated that the so-called protozoic disease of Posadas, Wernicke, and others, Busse's and Curtis's Saccharomycosis hominis, and Gilchrist's blastomycetic dermatitis are various manifestations of the same disease. The condition of the skin possesses constant clinical and histologic characteristics which separate it positively from all other skin diseases, particularly verrucose tuberculosis, carcinoma, and syphilis. The histologic features are: (1) A vast amount of "carcinomatoid" epithelial hyperplasia; (2) minute intraepithelial abscesses; (3) a granulomatous condition in the corium, characterized by masses of plasma-cells; minute abscesses, tuberculoid nodules, and giant-cells; (4) the presence of a spherical, capsulated, budding organism, particularly in the epidermal and subepidermal abscesses, but also distributed unevenly and in small numbers in epithelial masses and granulation tissue. Two histologic forms of the disease in the skin may be distinguished—the eosinophilous and the noneosinophilous, the former being associated with the mold type of the organism. The organisms isolated from the various cases differ in minor respects among themselves, but they are so closely related morphologically and biologically as to justify their inclusion in a common genus—Oidium; they are thus analogous in a pathogenetic sense to the fungi which cause actinomycosis, and to those causing trichophytosis. The variations among the organisms allow the recognition of 3 morphologic types: (1) Blastomycetoid or yeast-like; (2) oidium-like; and (3) hyphomycetoid. It is believed that oidiomycosis is an appropriate term for the conditions caused by the organisms, and oidiomycosis cutis for the condition as it occurs in the skin. The pathogenicity of the different organisms isolated varies. In the lower animals subcutaneous abscesses, septicemia, pulmonary consolidation, and pyemic foci have been induced by various methods of inoculation. Attempts to reproduce a typical lesion in man by inoculation of pure cultures have so far failed.

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[1] Jour. of Med. Research, VIII, 74, 1902.

[2] Jour. of Med. Research, VIII, 109, 1902.

[3] Jour. of Med. Research, VIII, 128, 1902.

[4] Jour. of Med. Research, VI, 373–546, 1901.

Two cases of **myoma sarcomatodes** are reported by Eising.[1]

A case of **chondrofibroma of the uterus,** containing cartilage, osteoid tissue, and bone, and thought to have resulted from metaplasia of fibrous connective tissue, rather than from aberrant embryonal rests, is reported by Kwarostonsky.[2]

Two cases of **lipoma of the uterus,** a very rare tumor, are reported by Merkel.[3] They resemble interstitial myomas and fibromyomas in their form, growth, seat, and influence on the shape of the uterus. It is believed that they result from proliferation of fatty tissue misplaced during fetal life, and not from metaplasia of smooth muscle-fibers.

A case of **primary epithelioma of the uvula,** occurring in a man aged 81 years, and involving the entire uvula, is reported by Oppenheimer.[4]

THE BLOOD AND DUCTLESS GLANDS.

The Changes Produced in the Hemolymph-glands of the Sheep and Goat by Splenectomy, Hemolytic Poisons, and Hemorrhage.— Warthin[5] has pursued his investigations of the hemolymph-glands (see the YEAR-BOOK for 1901, p. 403), studying especially the effects in the sheep and the goat of splenectomy, hemolytic poisons, and hemorrhage. The following are his conclusions: (1) After total splenectomy in the sheep there is no evidence of regeneration of the primitive spleen or of the new-formation of splenic tissue. (2) The structural changes following splenectomy are hyperplasia of existing lymphoid tissues, transformation of hemolymph-nodes into ordinary lymphatic glands, and a new-formation of hemolymph-nodes out of lobules of fat tissue, and a later proliferation of the red marrow. (3) There is no evidence of the formation of red blood-cells in the lymph-nodes after splenectomy. (4) The function of hemolysis is taken up first by the hemolymph-node, later by the ordinary lymphatic glands. (5) The hemolytic function of the hemolymph-nodes and hyperplastic lymph-glands exceeds that of the primitive spleen, causing an excessive destruction of red cells. The resulting anemia is later compensated for by an increased activity on the part of the bone-marrow. It would appear, therefore, that the removal of the spleen leads to an increased production or retention of some hemolytic agent usually disposed of by the spleen. The effect of this hemolytic agent is either to stimulate the phagocytes in the hemolymph-nodes to increased activity or so to change the red cells that they are more easily destroyed by these phagocytes. (6) The presence of great numbers of eosinophiles in the glands showing great destruction of red cells seems to point to some relationship between these cells. (7) The appearances described by Tizzoni, Winogradow, Eternod, Griffini, and Mosler, as occurring after splenectomy, are confirmed by this work, but given a different interpretation. Bayer's work upon the regeneration of the lymph-glands is also confirmed.

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[1] Jour. of Path. and Bact., VIII, 233, 1902.

[2] Ziegler's Beiträge, XXXII, 117, 1902.

[3] Ziegler's Beiträge, XXIX, 507, 1901.

[4] Med. Rec., Aug. 10, 1901.

[5] Jour. of Med. Research, VII, 435, 1902.

Granules in Leukocytes.—Michaelis and Wolff,[1] using a modified Romanowski stain, were enabled to demonstrate granules in a certain proportion of white blood-corpuscles previously considered granule-free—lymphocytes, Ehrlich's large uninuclear leukocytes, and transitional forms. It is believed that, like the morphologic characteristics which are not always sufficient to enable one to distinguish the lymphocytes from the large uninuclear cells, the granules are of little differential diagnostic value, and that there is no well-founded basis for distinguishing between cells that are morphologically alike and that possess similar granules. Hesse,[2] from a very careful study, concludes that Ehrlich's view relative to the specificity of the granules of the leukocytes is not tenable, and that there occur many transitional stages both in size and reaction to stains of the granules that render it extremely likely that transitional forms of the leukocytes themselves occur. These changes are believed to be expressions of functions of the cells. Inasmuch, then, as it is impossible to classify the leukocytes on a microchemic basis, it is suggested that they should be viewed as a single species of cell susceptible of extreme lability and alterations of tension, and possessed of a variety of functions, in which especially the granules are concerned. It is believed that the granules are structural constituents of the cells and that they are important factors in resorption, assimilation, and secretion.

Concerning Blood Degeneration and Regeneration.—Heinz[3] describes the morphologic changes and the fate of the red blood-cells in the 5 classes of vertebrates following injections with different varieties of blood-poisons (anilin, antifebrin, phenacetin, etc.). After a certain time the blood-corpuscles disappear completely from the circulatory stream, and their detritus is found, in the cases of the rabbit, chicken, frog and lizard, in the liver, spleen, and bone-marrow; in the case of the fish, in which the deposition of granular material goes on much more slowly, the blood-derivatives are found almost only in the spleen. He discusses also the regeneration of the red cells, which goes hand in hand with the destruction, its rapidity depending upon the poison used. In the cases of the first 4 groups the corpuscles are formed in the bone-marrow, and chiefly by the erythroblastic tissue of the marrow; in the cases of the fishes, regeneration takes place in the cephalic accessory kidneys. Reparation is rapid in the chicken, quite slow in the frog and fish.

Behavior of Leukocytosis in Pneumococcic Diseases of Rabbit and Man.—Williamson[4] infected rabbits subcutaneously with small doses of highly virulent pneumococci. A short time after the infection there occurred a considerable increase in the number of white corpuscles, followed later by a fall in the number, the diminution going far below the original count. The leukocytic curve varied greatly in the different experiments, and the leukocytosis lasted but a short time, on account of the very virulent cultures used in all cases. The hypoleukocytosis sank sometimes to as low a point as 1000. There seemed to be no relation between the hyperleukocytosis, the hypoleukocytosis, and the number of leukocytes existing before the infection. The behavior of the leukocytes is in no way connected with the clinical picture or the duration of the disease. In other words, it has no noticeable influence on the course of illness, and on that account Williamson doubts the importance of the leukocytes from the standpoint of being protecting agents for the organism. A few hours (3 to 5) after the infection pneumococci make their appearance in the blood. A well-marked connection exists between the appearance of the pneumococci septicemia in the rabbit and the hypoleukocytosis; shortly after the pneumococci appear in the circulatory stream the leukocytic curve declines. This is analogous to the behavior of the leukocytic curve in the fatally ending cases of pneumonia in man. The total absence of hyperleukocytosis in many of the latter cases is believed by the author to be due to a profuseness of bacteria in the blood.

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[1] Virchow's Archiv, CLXVII, 151, 1902. [2] Virchow's Archiv, CLXVII, 231, 1902.
[3] Ziegler's Beiträge, 1901, XXIX, p. 299. [4] Ziegler's Beiträge, XXIX, p. 41.

Blood-platelets.—Deetjen[1] states that the significance of the blood-platelets has long been in dispute, that some writers look upon them as degeneration products of the erythrocytes and the leukocytes, and others as the consequence of metabolic activity. He describes a new method of investigating them that leads him to view them as cells *sui generis*, inasmuch as he has been able to convince himself that they consist of a nucleus and a cell-body and have ameboid movement. His method consists of examining the platelets on a specially prepared agar mixture. This consists of a 1 % solution of agar, to which are added 0.6 % NaCl, 6 to 8 % of a 10 % solution of $NaPO_3$, and 5 % of a 10 % solution of K_2HPO_4. The phosphate salts are necessary to preserve the vitality of the platelets. A small amount of this agar mixture is smeared on a glass slide, permitted to harden, whereupon a narrow strip 2 mm. wide is cut out. Blood is then allowed to flow into this space and immediately covered with a cover-glass. It is fixed with 1 % osmic acid introduced under the cover-glass, after which the cover-glass is raised, washed, and stained. Hirschfeld,[2] from the result of his investigations, believes that without doubt the blood-platelets result from the erythrocytes. A small number of the erythrocytes only give rise to them, and in these the platelets result from the development of an endoglobular platelet (in some cases of more that one) which occupies the central depression of the erythrocyte. Ultimately these endoglobular platelets leave the erythrocytes through one (in case there be several, through several) opening in the enveloping membrane of the erythrocyte, and thus they become free platelets. It is believed also that similar formations result from the leukocytes, but that these

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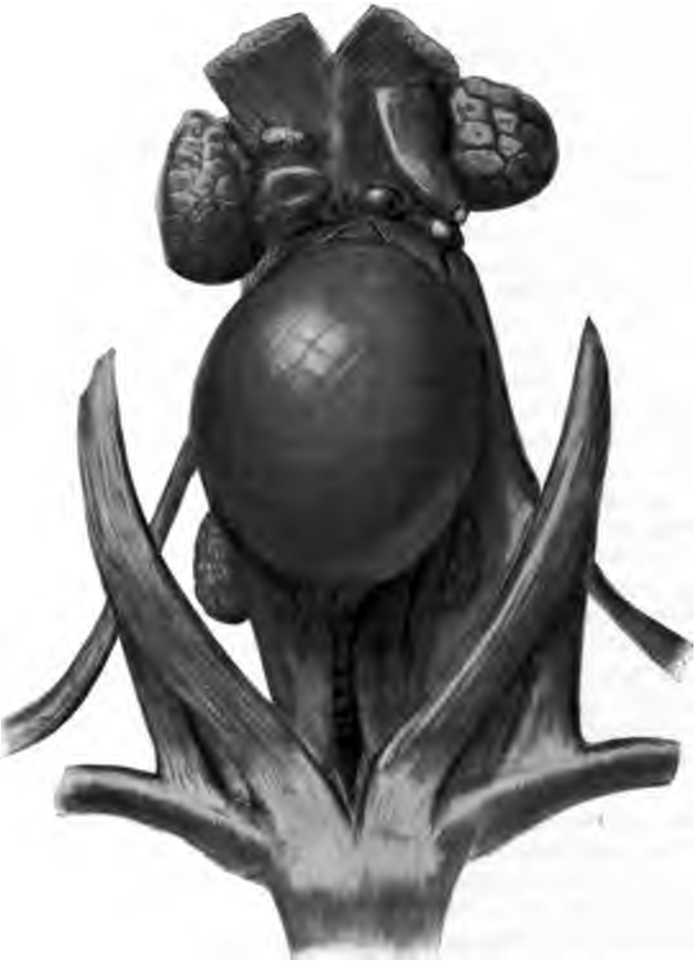
[1] Virchow's Archiv, CLXIV, 239, 1901. [2] Virchow's Archiv, CXLVI, 195, 1901.

PLATE 2.



The cyst with the sternohyoid muscles is turned upward on its attachment to the hyoid bone; the isthmus of the thyroid is exposed, showing a thin median strand of muscle-fibers which run upward toward the hyoid (Robinson, in Johns Hopkins Hosp. Bull., April, 1902).

are rare in normal blood, more common in leukemic blood. Hirschfeld, who has used Deetjen's method, to some extent concurs in the opinion of its utility, stating that it enables one to see many more platelets than ordinarily, and that it shows that the platelets possess ameboid movement (slow movement, however, in contradistinction to Deetjen, who believes the movement active). Hirschfeld, however, was unable satisfactorily to assure himself that the platelets possess a nucleus and cell protoplasm. Similar results were attained by Kopsch.[1]

The Blood and Bone-marrow in Chronic Arsenical Poisoning, with Especial Reference to Cutaneous Pigmentation.—Muir[2] states that the blood of arsenical patients having deep cutaneous pigmentation is decidedly richer in red cells and hemoglobin than the blood of similar, slightly pigmented or unpigmented patients; that this disproves the almost universally accepted hypothesis that the pigmentation is due to destruction of hemoglobin or red cells and is evidence for thinking that the melanin of the skin is an important element in the production of hemoglobin; that in human beings arsenic causes a decided erythroblastic reaction in the marrow of the long bones; that a slighter but distinct leukoblastic reaction also occurs which may be in part, or even wholly, due to accompanying tuberculosis; and that the erythroblastic function of the marrow is more easily stimulated in the presence of a large store of cutaneous pigment.

The Branchiogenic Organs of Man.—Erdheim[3] discusses the histology and histogenesis of the parathyroid glands of man, of which there are two on both sides of the thyroid, and he refers to the moot questions whether or not they are remnants of embryonal thyroid tissue that has not reached mature development, and whether or not they are capable of acting vicariously for the thyroid. Recent studies in cases of thyroid aplasia in which parathyroids were present show that the latter are not aberrant portions of thyroid, and that they are never transformed into thyroid tissue. They are probably formed from the third and fourth visceral clefts, those from the lower eventually coming to be the higher. The parathyroids, especially the lower, frequently are found cystic. The cysts of the upper pair originate either from the fourth visceral cleft or from the lateral thyroidal area; those of the lower, from the third visceral cleft. In the case described by Erdheim the cysts of the upper parathyroid bodies did not contain any thyroid tissue, probably because they were formed from the remains of the fourth visceral arch, and not from the lateral thyroidal area. In cases of total thyroidal aplasia the lateral thyroidal areas are probably as aplastic as the mesial. **A cyst originating from the ductus thyreoglossus** is reported by Robinson[4] (Plate 2). An unusual case of **branchial cyst the wall of which contained a small angioma** is reported by Coplin,[5] who recently discussed fully the pathology of branchial cysts.[6]

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[1] Anatom. Anzeiger, No. 21, 1901. [2] Jour. of Path. and Bact., vii, 420, 1901.

[3] Wien. klin. Woch., Oct. 10, 1901.

[4] Johns Hopkins Hosp. Bull., xiii, 81, 1902.

[5] Phila. Med. Jour., ix, 760, 1902.

[6] Proc. Path. Soc. of Phila., N. S., iv, 109, 1901.

A case of thrombosis of the central vein of the right adrenal with engorgement and necrosis (hemorrhagic infarction) is reported by Woolley.[1] The origin of the thrombus could not be made out; it was probably marantic in origin, judging from its structure.

Concerning the Parathyroid Glands.—Benjamins[2] concludes from his own observations on man, and the experimental studies of other investigators, that the parathyroid glands in animals are possessed of a physiologic importance of which very little is known. They have an independent embryologic origin, arising probably from the fourth branchial cleft. They occur constantly in pairs bilaterally and usually in definite positions. Their histologic structure differs at all times from that of the thyroid. The colloid substance is an accidental product. They are unaffected by regressive and progressive changes going on in the thyroid; if changes do occur, they are traceable to mechanical disturbance. Malignant tumors may have their origin in one of them. Parathyroid extirpation produces symptoms of an acute illness and death, which may be warded off by feeding the animals with parathyroid substance.

Compensatory Hypertrophy of the Suprarenals.—Simmonds[3] believes that atrophy or disease of one suprarenal may be compensated for by hypertrophy of the other. In addition to one case previously recorded, he reports 3 in which this was found. He states that one is justified in speaking of a compensatory hypertrophy in cases in which, with an atrophic adrenal, the other organ weighs more than 10 grams.

THE CIRCULATORY SYSTEM.

Fatty Degeneration of the Myocardium.—Cowan,[4] from an exhaustive study of the subject, concludes that fatty degeneration of the myocardium is of common occurrence. While marked cases give notable evidence of the change on macroscopic examination, in a majority of the cases it can be diagnosed with certainty only on examining sections which have been fixed in osmic acid. Three forms are met: In one the fat is irregularly distributed throughout the muscle, occurring especially in those parts which are relatively ill supplied with blood; in the second, the change is diffuse, and is produced by a toxic condition of the blood-stream; intermediate cases may be met in which both causes are present; the third form is due to local toxic action. So far as the microscopic evidence goes the condition is a true degeneration of the muscle-cell, and, besides the presence of fat, other changes are also found in the cells, involving the destruction of fibril bundles, and consequently leaving a marked impairment of contractile power. If the cause is remediable, the degeneration may be recovered from. The causes of fatty degeneration of the myocardium may be classified as follows: (1) A relatively insufficient supply of blood is afforded to the part (*a*) from abnormal qualitative changes in the blood—posthemorrhagic anemia, pernicious anemia, etc.; and (*b*) from local conditions—coronary obstruction or special stress on particular parts of the heart. (2) A toxic condition is present (*a*) in the blood-stream—phosphorus, arsenical, and chloroform poisoning, alcoholism, uremia, etc.; and (*b*) in the lymph-channels of the heart—pericarditis. (3) A combination of these causes may be present. In fatty infiltration it seems possible that the wandering fat may be deposited in the adjacent muscle-cells.

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[1] Jour. of Med. Research, VII, 231, 1902.
[2] Ziegler's Beiträge, 1902, XXXI, p. 142.
[3] Centralbl. f. allg. Path. u. path. Anat., 1902, XIII, No. 3.
[4] Jour. of Path. and Bact., VIII, 177, 1902.

Mechanical and Microtechnical Factors Concerned in Producing Segmentation and Fragmentation of the Myocardium.—Buhlig[1] details the results of his investigations undertaken to determine in how far the microscopic appearances designated "segmentation and fragmentation of the myocardium" are the result of artificial conditions. In this preliminary note he expresses the conviction that most of the recorded examples of segmentation and fragmentation of the myocardium are artefacts; but he does not make the assertion that all such cases are thus to be disposed of, though he suggests that further knowledge may lead him to conclude that a diagnosis of cardiac segmentation and fragmentation based upon the microscopic appearance of sections is erroneous in all cases. The factors of primary importance in producing the artefact are imperfections of the knife-edge, and the direction of the section—it having been repeatedly found that one direction of section (the pull of the knife through the section at an angle approximating 30 degrees with the long axis of the heart-fibers) was more favorable for the production of microscopic fracture than any other. Factors probably of secondary importance are reagents used in treating the tissue; the nature and consistency of the embedding mass; the knife and object vibration; the knife tilt; the thickness of the section; and the knife slant.

A Contribution to the Histopathology of the Typhoid Heart.—Drago[2] examined the hearts of 2 typhoid fever patients and found considerable overdistention of all the blood-vessels leading at some points to large lacerations and extensive hemorrhages. The intermuscular connective tissue showed lymphoid infiltration. Some of the muscle-fibers had disappeared, others were shrunken and disorganized. Drago considers the process one of simple atrophy in which the muscle-fibers become at first slightly cloudy, later deformed, reduced in size, and ultimately disappear. The muscle-nuclei disappear in a similar manner, but there is no connection between the two.

THE RESPIRATORY SYSTEM.

Disorders of the Lymphatics of the Lungs in Chronic Bronchitis.—Watanabe[3] gives the results of his investigations of 32 cases of chronic bronchitis of varying type and severity with particular reference to the changes in the lymphatic vessels. He believes that almost regularly in chronic bronchitis the lymph-vessels are involved in the inflammatory process—the finer vessels in the wall of the bronchi to a greater extent than the larger vessels. A marked implication can be seen even with the unaided eye—appearing as fine gray streaks and bands that form a network and that are especially observable at the points of crossing. The inflammatory process in the lymph-vessels decreases in intensity from the periphery toward the hilum of the lung; it is almost always acute, though chronic alterations have been seen. The lymphangitis is generally less marked in cases of uncomplicated bronchitis than it is in cases of bronchitis complicated with other affections, especially the common bronchopneumonia.

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[1] Jour. of Med. Research, VII, 428, 1902.

[2] Ziegler's Beiträge, XXIX, p. 176.

[3] Virchow's Archiv, CLXV, 80, 1902.

Fibrinous Bronchitis.—Bettman[1] reports a case of fibrinous bronchitis occurring in a woman aged 22 years, and he discusses in detail the features of all cases reported in the literature, which he divides into 9 groups: (1) Chronic bronchitis with expectoration of branching casts of the bronchial tree—23 cases; (2) acute bronchitis with expectoration of branching casts of the bronchial tree—15 cases; (3) cases in which branching casts were not expectorated, but were found in the bronchi at the autopsy—6 cases; (4) cases in which the casts expectorated showed no dichotomous branching—11 cases; (5) expectoration of branching casts in organic disease of the heart—10 cases; (6) expectoration of branching casts in pulmonary tuberculosis—14 cases; (7) expectoration of small casts, often nonbranching, in association with asthma—5 cases; (8) formation of casts in the bronchi in association with pulmonary edema following thoracentesis—4 cases; and (9) cases whose classification is doubtful, because of incomplete reports—6 cases. A critical and detailed analysis of the cases of each group is given.

The Pathogenesis of Bronchial Asthma.—Benedetto de Luca,[2] after discussing the various theories of bronchial asthma, says that in order to produce asthmatic attacks the reactive power of certain bulbar centers, especially the vasomotor, must be excited—the attacks themselves depending essentially on the special excitability of certain bulbar centers to various stimuli. Usually asthma attacks neuropaths whose nerve-centers are very irritable, but nonneuropaths may be attacked. In the neuropaths the asthmatic attack may be the clinical equivalent of some other neurosis. The essential pathogenetic fact in a fit of asthma is the narrowing of the bronchial caliber, caused partly by circulatory disturbance in the mucous membrane and partly by spasm in the muscles of the bronchial walls. In a complete attack both conditions must be present. The diaphragmatic spasm is a secondary result of the dyspnea, and must be classed with the similar spasm of the other accessory muscles of respiration. The bronchial secretion does not play any marked part in the pathogenesis of asthma, and the same applies to the spirals of Curschmann and the Charcot-Leyden crystals.

Bone Formation in the Lungs.—Pollack[3] gives the details of his study of bone formation in the lungs, stating that he has found in about

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[1] Am. Jour. Med. Sci., cxxiii, 304, 1902.

[2] Rif. Med., Dec. 6, 1901.

[3] Virchow's Archiv, CLXV, 129, 1902.

18 % of his necropsy subjects small, yellowish areas of bony hardness, and rarely larger than a pea, that contain smaller or larger amounts of true bone These, which consist not only of the hard tissue of bone, but also of medullary substance, result from metaplasia of new-formed cicatricial connective tissue, usually about calcified, necrotic foci. Advanced life and obsolescent tuberculosis are believed to be related etiologically to this bone formation, which, however, has nothing to do with true bone-tumor formation. It is believed also that the long well-known and very common, whitish, very hard (cartilaginous) foci in the lungs represent an early stage of this bone formation. Pollack states also that he has found in calcareous bronchial glands true bone, independently of bone formation in the lungs.

Concerning the Occurrence of Angiosclerosis in the Pulmonary Circulation.—Bruening[1] calls attention to the sclerotic processes occurring in the pulmonary circulation. He reports 21 observations, which show that it occurs oftenest and is most marked in the arteries of the lung, less marked in their veins; occasionally the bronchial arteries are affected, but never the bronchial veins. It is not as rare, therefore, as has been considered; it occurs especially in those cases in which the pulmonary circulation is interfered with, in pathologic states of the respiratory organs, and in combination with general arteriosclerosis. Chronic diseases, circulatory disturbances, and inflammatory processes in the vessel-walls and their surroundings are of as much importance in the production of pulmonary sclerosis as in sclerosis of the general circulation.

The Absorptive Power of the Pleura.—Grober[2] publishes the results of his extensive examinations concerning the anatomic structure and the absorptive power of the normal and the pathologic pleura, stating that the pleural cavity is connected directly through stomata with the lymph-vessels. The healthy pleura is capable of absorbing foreign bodies and fluids; every animal absorbing a definite quantity in a definite time, and this quantity corresponds to the size of the animal. Absorption is carried on by diffusion and osmosis, and by the respiratory movements, either through the pumping system of the intercostal lymph-vessels, the pressure of the lung against the thoracic wall, or the rubbing of the two surfaces of the pleura against each other. The absorptive power of the inflamed pleura is considerably diminished. The healthy pleura is capable of absorbing certain quantities of living micro-organisms and their toxins without harm to itself, thus making them innocuous for the organism. The inflamed pleura does not possess this property.

THE DIGESTIVE SYSTEM.

The Pathogenesis of Chronic Ulcer of the Stomach.—W. van Yzeren[3] discusses at length the peristaltic movements of the stomach, the tone of the stomach, the pressure within the stomach, the influence of the vagus on the stomach (excitation and section of the nerve), the consequence of vagotomy below the diaphragm, the anatomy of ulcer of the stomach produced by vagotomy in the rabbit, the time, place, number, manifestations, healing, prophylaxis, and genesis of ulcers of the stomach that develop after vagotomy, and the genesis of ulcer of the stomach in man. He points out the marked resemblance in the manifestations of ulcer of the stomach in vagotomized animals and in man—resemblances in anatomic appearances, number, situation of the ulcers, chronicity or intermittence, gastric cramp and intermission of the symptoms, the presence of perchlorhydria and the rare occurrence of hyperacidity, the immediate cessation of the symptoms and the rapid cure of the ulcer following gastroenterostomy and pyloroplasty, etc. From these he concludes that the genesis of ulcer of the stomach in vagotomized animals and of most cases of ulcer of the stomach in man is the same; and after some discussion, especially with reference to the effects of vagotomy, he concludes that the ulcers in both cases are due to spasm of the muscular wall of the stomach. In most cases the muscular cramps cannot be diagnosed, but in some cases their presence may be inferred by palpating a hard contracted pylorus and from the intermittent retention of the gastric contents. It is stated that whether or not disturbance of the vagus be found in the human subject suffering with chronic round ulcer of the stomach, it is certain that in most cases the ulcer arises in consequence of muscular spasm, and that this spasm is the cause of the nonhealing of the ulcer. To relieve the spasm and to promote healing of the ulcer a new operation—extramucous division of the pylorus—is proposed.

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[1] Ziegler's Beiträge, 1901, xxx, p. 457. [2] Ziegler's Beiträge, 1901 xxx, p. 267.
[3] Zeitschr. f. klin. Med., xliii, 181-224, 1901.

Primary Tuberculosis of the Liver.—Frank,[1] from a study of the literature and the investigation of a personal case, concludes that primary tuberculosis of the liver, though rare, may occur; that the infection may take place by way of the intestine and the portal circulation, the bacilli finding entrance through an ulcer in the bowel and leaving no trace of entrance into the body; that tuberculosis of the liver may infect the other abdominal viscera and also the peritoneum secondarily; that the process may cause a great increase of the connective tissue in the liver (interstitial hepatitis); and that the disease results fatally within 12 months.

A case of ulcer of the stomach caused by the diphtheria bacillus is reported by Stokes.[2]

Cirrhosis of the Liver with Multiple Adenomas.—Fraser,[3] reporting a case, reviews also the literature. He believes that primarily there occurs a hyperplasia of the connective tissue of the liver which at first is loose in character, but which later contracts and leads in some places to destruction of the liver-cells, in other places to compensatory hypertrophy. The latter is believed to occur in places where the connective tissue is not very dense, and where there is little resistance to growth, as on the surface of the organ. The hypertrophic masses are undoubtedly adenomas, and these are believed to originate in part from the epithelium of the bile-ducts, in part from the liver-cells. A case of **primary adenocarcinoma of the liver with widespread interlobular cirrhosis** is reported by Travis.[1] The new-growth had invaded both the portal and the hepatic veins, and filled the pulmonary arteries, giving rise to metastatic nodules in the lungs. Transitions from liver cords to tumor cells were demonstrable. It is thought that the tumor possibly resulted from progressive atypical growth of hyperplastic masses produced coincidently with the cirrhosis by a reparatory and regenerative process, after extensive destruction of the liver by some unknown poisonous agent.

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[1] Am. Jour. Med. Sci., CXXIII, 630, 1902.
[2] Johns Hopkins Hosp. Bull., XV, 209, 1901.
[3] Virchow's Archiv, CLXV, 540, 1901.

Acute Atrophy of the Liver and Multiple Nodular Hyperplasia.—Barbacci[2] reports the following case: A 45-year-old woman who was markedly emaciated and jaundiced died as the result of a double exudative serofibrinous pleurisy, associated with tuberculosis of the lungs, hypertrophy and infarction of the spleen, acute nephritis, gastroduodenal and biliary catarrh, ascites, and especially with a noteworthy lesion of the liver. This organ was of normal size; its surface form was greatly changed through the existence of large yellowish-white nodes, some of which were found scattered through the organ. On section they were soft, like liver-tissue. The internodular tissue was dark red, smooth, tough and spongy. No metastases were seen anywhere. Microscopically the internodular tissue was found to consist of fibrous connective tissue, rich in cells; scattered through it and lying in depressions were seen blood, remnants of liver parenchyma, many newly formed biliary canals, and blood-vessels; but little hepatic tissue remains, and most of this is in a state of degeneration. The nodes are made up of newly formed liver-cells separated from each other by large quantities of connective tissue, but the arrangement is not typical of hepatic lobulation. Nearly all the cells are in a greater or lesser state of fatty degeneration and necrosis. The author discusses the nature of the condition with especial reference to adenomatous growth, and sifts the literature for similar cases. His own case he believes to be one of parenchymatous degeneration terminating in nodular hyperplasia.

Primary Carcinoma of the Liver.—Eggel[3] has collected 163 cases of primary carcinoma of the liver from literature, and compared them with one another from various points of view. He reports a case of his own, giving a minute microscopic description. Inasmuch as atrophic cirrhosis and carcinoma are so frequently met together, the author believes that cirrhosis is to be looked upon as the primary process, the carcinoma resulting from proliferation of epithelial cells which have been displaced from their normal situations and transferred to the connective tissue either through developmental, traumatic, or inflammatory processes.

A large actinomycoma of the liver secondary to a circumscribed actinomycosis of the upper jaw is reported by Moodie.[4] The points of interest in the case are the large size of the actinomycoma of the liver as compared with the primary lesion, the possibility of demonstrating the hematogenetic origin of the process in the liver by the capsule-like formation, and the peculiarities of the actinomyces—no threads were found in the human body, but there were large quantities of granular masses and abundant bulbs, and threads were found in fresh cultures and there only.

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[1] Johns Hopkins Hosp. Bull., xv, 108, 1902.

[2] Ziegler's Beiträge, xxx, 1901, p. 49.

[3] Ziegler's Beiträge, 1901, xxx, p. 506.

[4] Jour. of Path. and Bact., VIII, 239, 1902.

Primary Carcinoma of the Bile-ducts.—Devic and Gallavardin[1] report a case of primary carcinoma of the common bile-duct and give a review of the previously recorded cases (55) of carcinoma of the hepatic duct and its branches, the cystic duct, and the common bile-duct (excluding the ampulla of Vater and the gall-bladder). Of 46 cases, 30 were males and 16 females. Most cases occurred between the ages of 50 and 66 years. Gall-stones were not so common as in carcinoma of the gall-bladder. In 53 cases the common duct was involved in 22, the junction of the hepatic, cystic, and common duct in 15, and the hepatic duct and its branches in 16. Metastases, practically confined to the liver, occurred in about 20 % of the cases. In 18 cases of carcinoma of the common duct in which mention is made of the condition of the gall-bladder, it was dilated in 17 and normal in 1; in 14 cases in which the tumor was at the junction of the common, hepatic, and cystic ducts, the gall-bladder was dilated in 7, normal in 3, and contracted in 4; in 7 cases in which the tumor was in the hepatic duct, the gall-bladder was dilated in 2, normal in 1, and contracted in 4. Of 37 cases in which the condition of the liver was noted, it was enlarged in 25 and normal or atrophied in 12. The following 4 clinical groups of cases are recognized: (1) The common form with enlarged liver and dilated gall-bladder, comprising almost all the cases of carcinoma of the common duct, and some of the cases of carcinoma of the hepatic duct. (2) An atypical form with or without enlargement of the liver, and atrophy of the gall-bladder, mostly cases of carcinoma of the hepatic duct. (3) A form simulating hypertrophic biliary cirrhosis—with enlarged liver and spleen. (4) A form without jaundice—rare, but one case having been reported. The disease has to be differentiated from hypertrophic cirrhosis of the liver, obstruction of the common duct by a calculus, an adjacent carcinomatous gland, a carcinoma of the ampulla of Vater, or a carcinoma of the head of the pancreas.

Nodular Cancer of the Liver Secondary to Cancer of the Ductus Communis Choledochus.—Sommer[2] reports a case in which the primary mass was found involving the common bile-duct, pancreatic duct, a portion of the duodenum and pancreas. The liver was very much enlarged and its surface was covered with nodules measuring from ½ to 4½ inches in diameter. The kidneys contained metastatic nodules.

Lesions of the Liver Caused by Distomata.—Blanchard[3] reports 2 cases of atrophic cirrhosis of the liver: the first in a sheep and due to Distoma lanceolatum, the second in a man and due to Distoma sinense. He describes their evolution from the ovum to the adult worm, their morphologic characteristics, the lesions they produce in the liver, and their manner of doing it.

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[1] Rev. de Méd., 1901, 557, 658, 839.

[2] N. Y. Med. Jour., Aug. 17, 1901.

[3] Bull. de l'Acad. de Méd., July 16, 1901.

The Causes and Varieties of Chronic Interstitial Pancreatitis.—Opie,[1] from an exhaustive study of 29 cases of chronic interstitial pancreatitis, concludes: (1) Chronic interstitial pancreatitis is slightly more frequent in males than in females; two-thirds of the total number of cases occur between the ages of 40 and 60 years. (2) The most frequent cause of chronic interstitial pancreatitis is obstruction of the duct of Wirsung, due to pancreatic calculi, to biliary calculi in the terminal part of the common bile-duct, or to carcinoma invading the head or body of the gland. Duct obstruction may be followed by the invasion of bacteria, which take part in the production of the resulting lesion. (3) Ascending infection of the unobstructed duct of Wirsung may follow an acute lesion of the duodenum or of the bile-passages, and may cause chronic inflammation. In cases which have given a history of long persistent vomiting, chronic diffuse pancreatitis may be found at the autopsy, and is probably the result of an ascending infection of the gland. (4) General or local tuberculosis is occasionally accompanied by chronic diffuse pancreatitis, affecting chiefly the interstitial tissue of the gland. (5) Chronic interstitial pancreatitis is not infrequently dependent upon the same etiologic factors, notably alcohol, which produce cirrhosis of the liver, and in about one-fourth of the cases the two lesions are associated. (6) Following duct and ascending infection the lesion affects principally the interlobular tissue, only secondarily invading the lobular tissue, and sparing the islands of Langerhans. Diabetes results only when the lesion is far advanced. (7) Accompanying the so-called atrophic or Laennec's cirrhosis of the liver, the pancreas is at times the seat of a diffuse chronic inflammation characterized by diffuse proliferation of the interacinar tissue which invades the islands of Langerhans; a similar lesion accompanies the hyaline degeneration of the islands of Langerhans and the condition known as hemachromatosis. (8) Interacinar pancreatitis is usually accompanied by diabetes mellitus. When diabetes is absent, the lesion is of such slight intensity that the islands of Langerhans are little implicated.

Degeneration of the Islands of Langerhans of the Pancreas in Diabetes Mellitus.—Wright and Joslin,[2] examining the pancreas from 9 cases of diabetes, found in 2 of the cases hyaline changes in the islands of Langerhans like those found by Opie. In the other 7 cases definite lesions were found in the pancreas in but one, and they consisted of an exudate of fibrin and leukocytes in the connective-tissue septa, the islands of Langerhans being not affected. It is believed that the occurrence of marked pathologic changes in 2 of the 9 cases is in favor of the hypothesis that lesions of these structures are important factors in the pathology of diabetes mellitus, but whether marked lesions of the islands of Langerhans will always be found to be associated with glycosuria must be determined by further observations.

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[1] Am. Jour. Med. Sci., cxxiii, 945, 1902.

[2] Jour. of Med. Research, vi, 360, 1901.

The Pathogenesis of Cysts of the Pancreas.—Lazarus,[1] in consequence of considerable experimental and other study, states that, according to their pathogenesis, pancreas cysts may be divided into two principal groups. To the first group belong those cystic formations derived from gland-ducts and acini. These may result in one of three ways: (1) Through proliferation—the cystoma glandulare proliferum, the most common form according to the statistics of operative cases. (2) Through retention—retention cysts, which result from the principal duct of the pancreas or its ramifications, such as the acini, in consequence of mechanical obstruction to discharge of the pancreatic juice. The most frequent cause is chronic interstitial pancreatitis. This genetic relationship indicates the etiologic identity of the two processes. (3) Through degeneration—lobular softening cysts—which may follow acute pancreatitis that occurs in consequence of acute infective diseases. To the second group belong pseudocysts (cystoids) which have no epithelial lining. These also may result in one of three ways: (1) Through softening in consequence of regressive metamorphoses in tumors or of foci of fat necrosis; (2) through autodigestion—those traumatic cysts in which, in consequence of indurative processes in the neighborhood of the hematoma, resorption was interfered with and retention of secretion occurred; (3) through the extravasation of blood and pancreatic juice in the bursa omentalisin consequence of rupture of the pancreas with reactive inflammatoiy encapsulation—hematoma pancreatis et bursæ omentalis.

The Pancreas and Tuberculosis.—Italia[2] gives the results of his study to the interaction of the pancreas and the tubercle bacillus. Experimental inoculation on the lower animals determined that the pancreas reacts to tuberculous toxin by hyperplasia of the connective tissue and subsequent contraction. The nondetection of the tubercle bacillus in a number of pancreases infected with tuberculosis led to the supposition of a specific action of the pancreas and its secretion on the tubercle bacillus, and suggested a series of experiments to determine the truth or falsity of the supposition. Simultaneous inoculation of the tubercle bacillus and pancreatic infusion was found to result in inhibition of the growth of the tubercle bacillus (proved by control experiments). Pancreatic infusion was found to be curative of a local tuberculous focus, inducing marked phagocytosis, but it was without effect in generalized tuberculosis. Concentrated infusions of the pancreatic infusion were found to induce pronounced reaction at the site of inoculation, an effect that was not observed if weak dilutions were used. Small doses were found to increase body-weight, large doses to decrease body-weight. A 2 % solution of pancreatin was found to be more effective than pancreatic infusion. Trypsin caused marked destruction of tissue at the site of the inoculation and, in large doses, death from paralysis of the nerve-centers. The administration of pancreatic infusion to human beings was not followed by as good results as was the experimental administration of the infusion to lower animals.

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[1] Zeitschr. f. Heilkunde, xx, 165–268, 1901.

[2] Rif. Med., Mar., 1902.

Gas-formation in the Biliary Passages.—Stolz,[1] from an investigation of several cases and a study of the literature, concludes that so far two organisms have been proved to be causative factors of gas-formation in the organs—the one Fränkel's bacillus of gas phlegmon (Bacillus aerogenes capsulatus of Welch) and bacillus of the colon group (Bacterium coli commune and Bacillus lactis aerogenes). These organisms may reach the liver either by way of the blood or through infection of the bile-passages. The former is the more common with the Fränkel bacillus, the latter with the colon bacillus. It is believed also that the infection of the liver occurs during life, but that in none of the reported cases has it been definitely proved that the formation of gas occurred before death; it always was a postmortem finding. In consequence, therefore, it is thought unlikely that gas-formation in any organ occurs prior to death.

THE URINARY SYSTEM.

The Pathogenesis of the Edema of Bright's Disease.—Albu,[2] from a study of the literature and from the results of a number of experimental investigations conducted on rabbits, concludes that the theories of Bright, Bartels, and Cohnheim with regard to the pathogenesis of the edema of nephritis each contains an element of truth, and that the solution of the still disputed question is to be sought in a combination of all three theories. With regard to Bright's theory it seems to be beyond question that the increase in the watery constituents of the blood (the result of the loss of the solids, especially the albumin and the salts) favors the transudation of the serum through the blood-vessels. The hydremic plethora of Bartels also seems to be of considerable influence, particularly when it is associated with diminished excretion of water, such as obtains in cases of nephritis. In this case the blood-vessels are overfilled with fluid which forcibly endeavors to find its way out through all the avenues of escape at its command. Should the intestine and the different glands, such as the kidneys, the salivary and the tear glands, etc., prove insufficient, the water then finds its way out of the blood-vessels into the subcutaneous tissues and into the serous cavities. With regard to Cohnheim's theory, while there is no definite proof of inflammatory alterations with increased permeability of the blood-vessels of the skin and subcutaneous tissues, increased permeability of the walls of capillaries nevertheless seems to be a condition essential to the enormous transudation of serum from the blood-vessels. This is evidently the characteristic pathognomonic factor in the production of the edema, whereas the hydremia and the plethora are but predisposing factors.

Malignant Medullary Hypernephroma of the Kidney.—Ohlmacher[3] details the histologic and histogenetic features of an unusual hypernephroma of the kidney—a medullary hypernephroma (the great majority of hypernephromas being derived from the cortical cells of the adrenal). The following are given as the principal histogenetic features of the tumor and reasons for ascribing it to the medulla of the adrenal: (1) The disposition of the reticulum, which by anastomosis forms round, oval, or elongated alveolar spaces filled with the characteristic cells, is peculiar. The arrangement of this reticulum is precisely that of the adrenal medulla in the dog. (2) The character of the component cells, which are large and polyhedral, with large, faintly staining nuclei, and a scanty, poorly staining vacuolated cytoplasm, is identical with that of adrenal medullary cells. (3) The widening of the venules and blood-sinuses as deeper portions of the tumor are reached is similar to that shown by the adrenal medulla. (4) The presence of embryonic or immature medullary cells beneath the capsule of the tumor, and from here penetrating into the deeper portions, at the same time undergoing a transition, is like that seen in the developing normal adrenal. (5) Another of the interesting histogenetic features of this tumor concerns the occasional islands of cylindric cortical cells which take a distinct glomerular arrangement like that seen in the zona glomerulosa of the horse or dog and which are regarded by Kelly as indicating a reversion to a lower vertebrate type. (6) In the capsule of the tumor peculiar epithelial-clad tubules are occasionally seen which are regarded as remnants of the wolffian body. (7) The possibility of mistaking the histogenetic nature of the primary tumor from the structure of accessory nodules or from metastases is also indicated.

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[1] Virchow's Archiv, CLXV, 90, 1902.
[2] Virchow's Archiv, CLXVI, 87, 1902.
[3] Jour. of Med. Research, VII, 421, 1902.

Compensatory Hypertrophy of the Kidney.—To determine the pathologic changes in the kidney in cases of compensatory hypertrophy, Galeotti[1] and Villa Santa have performed unilateral nephrectomy on a number of dogs and rabbits with the following results: In the case of growing animals the main change consists in augmentation of the number of malphigian glomeruli; they retain their normal size. The number of convoluted tubules increases, but not the size of their cells. The lumen of the straight tubules dilates, owing most likely to the fact that a larger number of tubules terminate in them. Bellini's tubes are also dilated. In adult animals the glomeruli do not increase in number but they do in the size. The individual cells and the amount of secreting surface of the convoluted tubules increase in size, but not the number. The straight tubules remain unchanged, but Bellini's ducts dilate and become thinner. In both cases the hypertrophied kidney has a greater vascular filtrating surface, almost double that of the normal kidney; in young animals this is due to an increase in the number of glomeruli, in adults to an increase in surface space, of the individual glomerulus. In both cases there is an increase in secreting substances; in young animals this is due to a formation of new convoluted tubules without increase in the size of the cells, in adults the canals elongate and the cells hypertrophy. The straight tubules do not hypertrophy, thus proving that they have no secreting function.

The Association of Stone and Tumor of the Urinary Bladder.—Rosenow,[2] reporting a case of vesical calculus associated with carcinoma

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[1] Ziegler's Beiträge, 1902, xxxi, p. 121.
[2] Am. Jour. Med. Sci., cxxiii, 634, 1902.

of the bladder in a man aged 35 years, gives a summary of 44 similar cases reported in the literature. In 18 cases the stone was primary, in 8 the tumor was primary. It is not known which was primary in the other cases. The following arguments are given as favoring the view that the tumor increases the liability to the formation of calculus: (1) In 8 cases there is no question that the calculus was secondary to the tumor; (2) tumors, especially malignant tumors, are not infrequently incrusted with phosphatic material; and (3) villous growths are especially liable to cause calculi because a poorly nourished projecting villus, while yet attached or when torn off, may afford a nucleus, much the same as a foreign body, around which urinary salts are deposited. The following facts are given as indicating that vesical calculus is a factor in the causation of tumors of the bladder: (1) In over half of the cases it appears that the stone was primary; (2) inflammation of the urinary bladder caused by irritation other than that produced by a calculus has been found to cause an increased liability to new-growths; (3) tumors of the urinary bladder uncomplicated by stone occur more frequently in the lower segment; (4) the large number of cases in which the calculus was primary, and the fact that both calculus and tumor occur more frequently in the male sex and nearly at the same age, may be taken to indicate that the calculus was not without etiologic significance in the development of the tumor; and (5) since in 90 % to 95 % of primary carcinomas of the gall-bladder biliary calculi are present, analogy would lead us to expect a somewhat similar relation between stone and tumor in the urinary bladder. It is believed, then, that there is an etiologic relationship between calculus and tumor of the bladder, and that calculus seems to favor the development of tumor in a larger percentage of cases than tumor favors the development of stone.

Primary Squamous Epithelioma of the Renal Calices and Metaplasia of the Epithelium of the Calices, the Pelvis, and the Ureter.—Kischensky[1] reports a case of primary squamous epithelioma of the right kidney, beginning in the epithelium of its calices and pelvis, and extending from there throughout the parenchyma of the kidney, the liver, the neighboring diaphragm, peritoneum, and lymph-glands, and producing metastasis to the right knee-joint with involvement of the epiphysis and the lower part of the diaphysis of the femur. The epithelial pearls in the right kidney show considerable tendency to horny degeneration and secondarily to calcification; there was also well-marked epidermoid metaplasia of the mucous membrane of the pelvis, the calices, and the right ureter. The bladder and the left ureter were inflamed but without epithelial metaplasia.

The Fats and Allied Bodies in Chylous Urine.—Austin[2] gives the following as the results of his examination of 2 specimens of chylous urine: The first 500 cc. contained: total ether and alcoholic extracts 2.6173, fatty acids 0.005, cholesterin 0.015, oleic acid 0.1764, and palmitic and stearic acids 0.031. The second 500 cc. contained: total ether and alcoholic extracts 3.126, fatty acids 0.0032, cholesterin 0.0167, oleic acid 0.201, and palmitic and stearic acids 0.040.

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[1] Ziegler's Beiträge, 1901, xxx, p. 348.

[2] Jour. of Med. Research, vi, 366, 1901.

Monohydric magnesium phosphate, a hitherto undescribed urinary deposit, is described by Bradshaw.[1] It was found in the urine of a man with dilation of the stomach who had taken considerable doses of "powdered magnesia."

THE NERVOUS SYSTEM.

The Cerebrospinal Fluid in Meningitis.—Widal, Sicard, and others recently have upheld the importance from a diagnostic point of view of a microscopic examination of the cerebrospinal fluid in meningitis. Millian has supported the view that the discovery of unicellular leukocytes in the fluid of meningitis points to the tuberculous type of the disease, whereas polynuclear cells indicate an acute inflammation. Marcon-Mutzner[2] warns against the acceptance of this opinion in too absolute a fashion; he relates the history of a patient that died with symptoms of basal meningitis; fluid drawn by lumbar puncture contained polynuclear leukocytes exclusively, but at the necropsy the condition was found to be tuberculous. Thus the presence of polynuclear leukocytes is not conclusive evidence against tuberculosis; nor is the converse true, for the author relates a case proving that the presence of mononuclear cells is no proof of the presence of tuberculous meningitis. He also disagrees with the opinion that lumbar puncture is a harmless procedure. Wolff[3] states that the appearance of the cerebrospinal fluid, whether it be limpid, turbid, purulent, or contain much or little fibrin, permits the formulating of good rules for diagnostic purposes. In cerebrospinal meningitis the meningococcus, the pneumococcus, or the streptococcus may be isolated. The former is said to lose its virulence early and not to be found in many cases in which it is sought diligently. The tubercle bacillus is difficult of detection. It is suggested that in the future valuable information may be derived from a chemical study of the fluid, especially the albumoses. It is said that if the majority of the white cells in the fluid obtained by lumbar puncture are lymphocytes, the process is subacute or chronic; if, polynuclear, acute. The permeability of the meninges for substances like potassium iodid is of value in the diagnosis of tuberculous meningitis. While cryoscopy is not of great importance, hypotonia is common in acute and subacute cases, whereas in chronic cases the freezing-point is higher.

Congenital Porencephalus.—Shirres[4] reports the case of a woman, dying at the age of 76 years, whose mother was reported to have been frightened at the eighth month of pregnancy, and to have attempted abortion. The birth of the patient, however, occurred normally. Rigidity and paresis of the right arm and leg were noted about the second week. There was general backwardness of the child in walking and talking. At the age of 20 years she was to all purposes a normal woman, She was married twice but bore no children. At or about the age of 60 years deterioration of the mental faculties began. There were never any fits. There was kyphosis of the dorsal vertebras with slight dragging of the right foot, and some spasticity of the right arm and hand. The necropsy and subsequent careful study of the nervous system revealed a probable primary lesion of the middle cerebral artery (thrombosis), the occlusion taking place immediately after the perforating arteries from the basal ganglia are given off. This lesion had apparently occurred in the later months of fetal existence, and was due to rupture of fetal vessels in the placenta. The lesion was followed by atrophy and destruction of a large section of the cerebral cortex on the left side in an area supplied by the middle cerebral artery. The destruction was more extensive than was apparent at first sight, the cavity being in part filled in by the puckering and collapse of the surrounding tissue. The porencephalic cavity thus formed did not communicate with the lateral ventricles, but was bounded externally by the arachnoid, while the pia lined the sides and bottom of the cavity. In addition, there was a general diffuse atrophy of the cerebral convolutions of the left side (but these were not simplified), with a marked lessening of the large pyramidal cells and of the projection system of fibers, especially in what remained of the motor region. The association fibers were apparently normal. The right hemisphere was normal in shape and structure and revealed an increase in the large motor cells. The paper comprises details of a very careful study of the case and a thorough review of the literature.

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[1] Lancet, May 3, 1902.

[2] Arch. gén. de méd., vi, 1902; Brit. Med. Jour., Feb. 15, 1902.

[3] Gaz. hebd. de Méd. et de Chir., Sept. 8, 1901.

[4] Studies from the Royal Victoria Hospital of Montreal, vol. i, No. 2, 1901.

The Histology and Histogenesis of Internal Hemorrhagic Pachymeningitis.—Jores and Laurent[1] report a number of cases of so-called internal hemorrhagic pachymeningitis, and, scrutinizing these as well as others previously reported by themselves, they conclude that all cases reported under this name should be divided into 3 separate and distinct groups: (1) Cases in which the primary process is a subdural hemorrhage, the hemorrhage undergoing organization. This membrane superficially closely resembles that of pachymeningitis. The ultimate result is a fibroid thickening of the dura. (2) In a second group occurring especially after infectious diseases, the process begins with a deposition of fibrinous or fibrinohemorrhagic material on the dura; this also becomes organized, forming a connective-tissue membrane which is intimately attached to the dura, the exudative process usually extending deeper. (3) A third group is characterized by the formation and proliferation of a very vascular membrane, arising from the capillary layer of the dura; a tendency to small and large hemorrhages exists. The process is progressive, a return to normal conditions never taking place.

Cyst-formation in the Pineal Gland.—Barratt[2] details the results of his study of cyst-formation in the pineal gland, based upon the examination of 15 pineal glands obtained at necropsies occurring in asylum practice, and of 2 pineal glands obtained from healthy subjects killed by accident. In addition, comparison was made with the structure of the epiphysis of the calf, dog, cat, rabbit, and of ammocœtes. Cysts were found in 47 % (8 out of the 17) of the human pineal glands examined, and they were usually multiple. No definite relationship could be made out between the cystic change and age, sex, mental, or accompanying visceral disease. In all except 3 cases brain atrophy was present; in 1 of these 3 cases a single pineal cyst was found, and cysts were absent in the other two; in the remaining cases cystic change was present in 7 pineal glands out of 14. The cysts were not limited to any special locality of the glands. It is said that the cysts do not arise as well-formed diverticula of the pineal recess, and are not limited by a single layer of epithelial cells in contact with each other, resembling those lining the ventricles of the brain. In the formation of the pineal cyst disappearance of the proper cells of the gland occurs at the site of the cyst accompanied by an active transudation of fluid. At the same time hyperplasia of the neuroglia fibers all around the cyst takes place. In this way a neuroglial feltwork, often abundant, especially about the larger cysts, is formed, surrounding the cavity and constituting its wall. The proper cells of the pineal gland are present in this feltwork in small numbers and do not form a well-defined layer. No relation between the presence and amount of brain sand and cystic change was observed.

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[1] Ziegler's Beiträge, 1901, XXIX, p. 486.

[2] Jour. of Path. and Bact., VIII, 213, 1902.

The Spinal Cord Lesions in Pellagra.—Sandwith,[1] having had the opportunity to study the spinal cord lesions in 3 cases of pellagra poisoning, gives the following as the results: In the first case there was some degeneration in the posterior columns, as shown by Pal's method—a degenerate root entered at the third lumbar segment and was traced up to the dorsal region; the posterior median columns were unduly pale. In the second case well-marked degeneration of the posterior columns was present, evidently of root origin; there was also an increase of the connective tissue in the area of the affected roots, and some thickening of the walls of the arteries; the posterior roots themselves also showed marked degeneration in their extramedullary course. The third case revealed practically no deviations from the normal, except possibly some slight alteration in the medullated sheath of certain fibers. It is believed that the sclerosis of the posterior columns was of root origin, and that the increase of the connective tissue in the posterior columns was secondary to the degeneration of the roots. That the process was essentially chronic is shown by the fact that, although the posterior columns are extensively sclerosed, no degeneration could be revealed by Marchi's method.

The Pathology of Disseminated Sclerosis.—Shoyer[2] states his belief that the distribution of the lesions of disseminated sclerosis can be explained by the assumption that they are caused by a poisonous agent conveyed by a cerebrospinal fluid which finds entry along the fissure from the central canal and ventricles, along the nerve-roots, and at a point in each lateral margin of the cord. The poison is believed to be the result of perverted metabolism of nervous or other tissues.

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[1] Jour. of Path. and Bact., VII, 460, 1902.
[2] Jour. of Path. and Bact., VIII, 124, 1902.

Four cases of acute hemorrhagic meningomyelitis in cocker spaniels are reported by Brown and Ophuls.[1] The lesions consisted especially of hemorrhage about the cord (in the membranes) and of extensive hemorrhage, softening, necrosis, and degeneration of the cord. Bacteria could not be cultivated from any case, there was no evidence of interference with the blood-supply, and there was no history of traumatism. It is believed, therefore, that the lesions in these cases, as well as in some other cases of meningomyelitis, are due to toxemia.

Gliomas of Ependymal Origin.—Mallory[2] reports 3 gliomas of ependymal origin, two occurring in the fourth ventricle, and one over the coccyx.

Traumatism and Inflammation of the Brain.—Ehrnrooth[3] has made some experiments to determine whether cerebral inflammation can occur after injuries to the head that do not cause solution of continuity. He subjected rabbits to blows upon the head, and, before, simultaneously, and at various intervals afterward, injected pathogenic organisms—streptococci, staphylococci, and pneumococci—into the circulation. The results were that of the animals receiving injections of streptococci, 54.4 % developed infectious processes in the brain; of those in which the staphylococcus was employed, 56.3 %; and of the ones in which the pneumococcus was injected, 63.6 % reacted with inflammatory lesions. In addition, he reports autopsies on two human cases that illustrate the possibility of inflammation following upon injuries not causing solution of continuity. The injury brings about a *locus minoris resistentiæ* where any bacteria in the circulation may become localized. [This has an important medicolegal value.]

The Pathology of Poliencephalomyelitis.—Alice Hamilton,[4] reporting a case of the rare condition of poliencephalomyelitis, occurring in a girl aged 5½ years, reviews also the literature. The lesions in the case consisted of engorgement of the vessels of the gray and white matter throughout the cervical cord, medulla, pons, and crura; perivascular infiltration of round and oval cells in the gray matter and to a less extent in the white matter from the lower end of the decussation of the pyramidal tracts to the exit of the third nerve, this infiltration being more marked on the right than on the left side; degeneration of the multipolar cells of the anterior horn of the cervical cord, especially on the right side, of the cells of the lower third of the nucleus of the seventh nerve on the right, and of the lower third of the nucleus of the sixth nerve; edema and numerous hyaline bodies in the gray and white matter and in the perivascular spaces in the tegmentum of the pons at the level of the nucleus of the fourth nerve extending beyond the exit of the third nerve; engorgement of the vessels and edema of the brain; no capillary hemorrhages and no degeneration of the white tracts. The disease is believed to have been toxic in origin.

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[1] Jour. of Med. Research, VI, 344, 1901. [2] Jour. of Med. Research, VIII, 1, 1902.

[3] Till kännedomen om traumats betydelse för uppkomsten af infektiösa cerebralakommor. En klinisk-anatomisk och experimentell studie. Akademisk afhandlung. Helsingfors, 1901. Abstracted in Nord. Med. Arkiv., 1901, Bd. XXXIV.

[4] Jour. of Med. Research, VIII, 11, 1902.

Pathogenesis of Exophthalmic Goiter.—Tedeschi,[1] experimenting on dogs, finds that if the restiform bodies are injured anteriorly,—that is, just behind the acoustic tubercle,—most of the symptoms of exophthalmic goiter appear: namely, exophthalmos, enlargement of the palpebral fissure, tachycardia, generalized tremor, and sometimes polyuria, glycosuria, and abundant salivation. In rabbits the pupils dilate; in dogs the early dilation gradually lessens. The experiments were not decisive with regard to the thyroid. The restiform bodies were reached either by exposing the posterior extremity of the cerebellar vermis and raising it, or through the occipito-allantoid membrane and exposure of the commencement of the floor of the fourth ventricle. The lesions of the restiform bodies were produced by a small-bladed bistoury.

Chorea and Microorganisms.—Fornaca,[2] pointing out the association of chorea and microorganisms, states that he was able to demonstrate the presence of staphylococci in the cerebrospinal fluid of 2 cases of chorea. In a third case of chorea following erysipelas, streptococci were found in the blood, urine, and cerebrospinal fluid. Fornaca believes that at least a third of all cases of chorea are antedated by a febrile attack, especially scarlet fever, measles, and erysipelas, and that in all cases the cerebrospinal fluid should be examined.

MISCELLANEOUS.

Chronic Inflammation of the Serous Membranes (Multiple Progressive Hyaloserositis).—Nicholls,[3] writing on a somewhat rare form of chronic inflammation of the serous membranes—a condition that he designates multiple progressive hyaloserositis—reviews the literature (from which he collects 13 cases) and reports 2 personal observations. He directs particular attention to the etiology and pathogenesis of multiple hyaloserositis, to the association of cirrhosis of the liver and perihepatitis, and to the forms of peritonitis and their relationship to the "*Zuckergussleber.*" He states that there is an affection of the serous membranes—multiple progressive hyaloserositis—characterized by the formation of a dense fibrohyaline investment in certain regions, and of which two forms anatomically may be distinguished—the sporadic and the diffuse. The disease is primarily and essentially due to chronic inflammation, and it is almost certainly attributable to the action of microorganisms of low virulence. The exact nature of the microbic excitants is in many cases obscure, but cases undoubtedly exist that are due to the tubercle bacillus. Apart from tuberculosis, only one form—namely, chronic hyaline perihepatitis (*Zuckergussleber*)—gives rise to a clinical symptom sufficiently definite to render diagnosis possible. This form of perihepatitis may exist *per se*, but it is often secondary to chronic peritonitis, or, again, it may be part and parcel of a generalized affection of the serous membranes. The most important etiologic factors in chronic perihepatitis are, in order of frequency: acute and subacute hepatitis, chronic mediastinopericarditis, chronic peritonitis, and chronic pleuritis. Chronic hyaline perihepatitis, atrophic cirrhosis of the liver, chronic tuberculous peritonitis, and carcinoma of the peritoneum present many features in common; differential diagnosis, however, is possible in some cases. Four forms of hepatic manifestations are to be recognized: (1) Cirrhosis without perihepatitis; (2) chronic perihepatitis without cirrhosis (Curschmann's *Zuckergussleber*); (3) chronic perihepatitis with cirrhosis (Glissonian cirrhosis); and (4) cirrhosis with perihepatitis. To insure accuracy and to avoid misinterpretation the terms chronic pleurisy, pericarditis, perihepatitis, etc., should be discontinued in speaking of fibrous adhesions due to an inflammation past and gone, and should be restricted to those cases in which there is evidence of an active inflammation present at the time of observation.

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[1] Gaz. degli Osped., April 6, 1902; Brit. Med. Jour., June 7, 1902.

[2] Rif. Med., No. 74, 1901.

[3] Studies from the Royal Victoria Hospital, Montreal, vol. I, No. 3, 1902.

Filarial Lymphatic Varix.—Opie[1] discusses the discovery, the geographic distribution, and the life-history of Filaria bancrofti. He shows that 2 hosts are required to complete the cycle of its life-history. The embryo filaria are withdrawn from the human body by certain species of mosquitos; in their body they undergo a series of developmental changes. When blood containing embryo filaria is drawn into the stomach of the mosquito, the parasite loses its sheath, and by active movements makes its way through the stomach-wall into the thoracic muscles of the insect; here it acquires a digestive tract and other rudimentary organs. It then makes its way into the head of the mosquito, finally penetrating the probiscis. Here 2 worms are almost constantly seen together. He reports a case of filarial infection imported from the West Indies, and studied by him. The clinical condition referable to the parasite was the presence of a tumor-mass in the right groin, which, according to the patient's statement, had been for 8 years supposed to be a hernia. Autopsy demonstrated the existence of an immense plexus of enormously dilated lymphatic vessels lying in the abdomen and pelvis in front of the vertebral column. Continuous with this plexus was the distended varicose thoracic duct passing beside the aorta to the base of the neck. Dilated lymphatics held together by fibrous tissue followed both spermatic veins as far as the epididymis, and a similar mass of lymphatic vessels passed with the femoral vein and artery under Poupart's ligament and formed the mass which during life was mistaken for a hernia. An adult filaria occupied a dilated lymphatic vessel near the right epididymis, and a single embryo was found in a blood-vessel of the liver. The entire lymphatix varix was the seat of acute inflammation characterized by the accumulation of polynuclear leukocytes in the distended vessels, and in the intervening tissue. The lymphatic vessels were filled in great part with blood, and had everywhere undergone thrombosis. Bacteriologic examination demonstrated infection with Diplococcus lanceolatus; secondary infection of the serous cavities in contact with the varix had occurred, and death resulted with acute fibrinopurulent inflammation of the peritoneum, right pleura, and both tunicæ vaginales.

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[1] Am. Jour. Med. Sci., Sept., 1901.

Elastic Tissue in Cicatrices.—Pezzolini,[1] by means of a series of experiments, demonstrated that elastic fibers in cicatrices develop gradually from adult elastic fibers. He produced wounds of uniform size in dogs, permitting some of them to heal by cicatrization; others had skin grafted on them. After complete healing, specimens including the cicatrices and surrounding tissue were excised and examined, in stained condition, under the microscope. About the sixtieth to seventieth day new elastic fibers appeared, varying in numbers according to the age of the cicatrix. They appeared first at the lateral margins, their development in the center being much delayed, except in those cases in which adult connective tissue was grafted on, when the lateral and central growths were simultaneous. The divisions of the ends of the cut elastic fibers, and their development and extension into the connective tissue of the cicatrices, were observed.

The Endothelial Phagocytes of the Tonsillar Ring.—Goodale[2] states that in normal tonsils, both of man and of a variety of the lower animals, there are present large mononuclear phagocytes which appear to be derived from a proliferation of the endothelial cells of the reticulum. In hyperplasia of the tonsils there is a disproportionate increase in the proliferation of the endothelial cells of the reticulum, and the formation of phagocytes, without, however, a corresponding increase in the number of lymphoid cells or in the endothelial cells of the blood-vessels. In atrophy of the tonsils the endothelial cells of the follicles are seen first to diminish in number, and endothelial phagocytes are correspondingly few, while the lymphoid cells persist relatively longer. In acute inflammation the characteristic feature of the process consists in a heightened proliferation of the endothelial cells of the blood-vessels, with swelling of their cytoplasm, together with an increased number of lymphoid cells. These changes are relatively more marked than the increased endothelial proliferation of the reticulum with follicles. When circumscribed abscess formation occurs in the follicles, an increased proliferation of the endothelial cells, both of the reticulum and of the capillaries, is observable in the vicinity. When an intense irritant acts upon the endothelial cells of the tonsil, degeneration and necrosis result in the immediate vicinity. At a greater distance proliferation appears to be excited in these cells. It seems, therefore, probable that proliferation of the endothelial cells of the reticulum, with a formation of phagocytes, is not necessarily dependent upon the influence of bacterial toxins for its production, but appears rather to stand in a definite relation to the size or activity of the organs in question. Proliferation, on the other hand, of the endothelial cells of the blood-vessels was found to occur only in association with other phenomena of inflammation.

The Function and Destiny of the Cells of Inflammatory Exudations and the Origin of the Mononuclear Cells.—Beattie,[3] in consequence of considerable experimental investigation, states that after an injection of bacteria into the abdominal cavity various cells appear in the exudation. In the early stages the polymorphonuclear leukocytes migrate from the vessels and appear in great numbers in the peritoneal effusion. These are found abundantly in from 6 to 48 or 54 hours after the injection. In nonfatal cases they diminish in from 48 to 60 hours, but in fatal cases the increase persists until the death of the animal. These leukocytes are the main bacterial phagocytes. The bacilli of tuberculosis and of leprosy seem to be exceptional, but this is probably explained by their slower growth and consequently less marked and later polymorphonuclear leukocytosis. During the time they are in excess there appears to be produced a substance injurious to the life, or at any rate to the activity, of the bacilli; but it is doubtful whether this is produced by these leukocytes or by the mononucleated cells, which are also in a state of great activity. The activity of the bacteria seems to be impaired before they are ingested by the leukocytes. These leukocytes are largely destroyed in the fluid, either as free cells or in the interior of other cells. The coarsely granular eosinophile cells, which are very numerous in the peritoneal fluid in healthy guinea-pigs, play no important part, at any rate in relation to the bacteria used during the investigations. The mononucleated phagocytes are found at all stages. They are most abundant from 36 hours onward. They are derived partly from the endothelium of serous membranes, of blood-vessels, or lymph-vessels, of lymph-sinuses, etc., partly from the large mononucleated leukocytes, and partly from the lymphoid tissue surrounding the vessels. They are ameboid and show, especially during their stages of greatest activity, a marked multiple vacuolation of their protoplasm, which is probably evidence of secretory action on the part of these cells, and not merely of a degenerative change. These cells are especially phagocytic to other cells. They may also ingest bacteria, and certain forms apparently more readily than others. The bacilli of tuberculosis and of leprosy are found in great numbers in these cells, and they appear actually to multiply in the cells. The ingested leukocytes undergo a gradual process of digestion by these mononucleated cells. These cells are largely destroyed in the peritoneal sac. None of the internal organs, so far as could be ascertained, appears to act as a storehouse for, or a destroyer of, these cells, with the exception possibly of the lymph-glands. Great numbers of the cells are always found on the omentum. These mononucleated phagocytes are the most important cells of inflammatory exudations. In cases of peritonitis the endothelium of the omentum furnishes a great number of these cells. The omentum must, therefore, be regarded as an important agent in protecting the individual from infection by way of the peritoneum. The presence of large numbers of these mononucleated cells, if they are actively functioning, in inflammatory exudations, must be regarded as a favorable sign. The plasma cells which Mallory suggests may in typhoid fever produce the antitoxin are believed to be the mononucleated cells which are derived from the large mononucleated leukocytes. They can and do act as phagocytes, but in the later stages of infection this phagocytic action is not seen, simply because there is no need for them to functionate in this way. They are not confined to any special form of inflammation, but are most numerous in cases of bacterial or of toxin infection.

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[1] Gaz. degli Osped., Dec. 22, 1901.

[2] Jour. of Med. Research, VII, 394, 1902.

[3] Jour. of Path. and Bact., VIII, 129, 1902.

The Endothelium in Inflammation.—After discussing the embryologic nature and anatomic classification of endothelium, LeCount[1] states that the changes which the endothelial cells may undergo in inflammation comprise all the retrogressive processes except that of sudden actual destruction, and all the phenomena included under the terms regeneration or proliferation except those of tumor growth. It must also be accepted that the behavior of these cells in inflammation, their proneness to resist, or their likelihood to suffer, great damage is quite in proportion to their differentiation. Endothelial cells, during inflammation, absorb nonmotile bacteria, acting as phagocytes; they also digest red blood-corpuscles, plasma cells, and leukocytes. While doing this the nucleus and cell-body become larger; and when filled entirely with bacteria, desquamate. Proliferation of the endothelium plays an important part in the formation of fibrin during acute serous inflammation. The new cells may form an unbroken row between the fibrin and the serous coat, or the fibrin may be almost surrounded by newly produced endothelium. Fibrin ferment arises, in part at least, from the destruction of endothelium cells. When the endothelial cells of serous membranes do not suffer necrosis, they undergo a lively proliferation. Many observations have been made of the formation of giant-cells by endothelium in both blood- and lymph-channels and in serous membranes. In some inflammations toxins have caused multiplication of endothelium lining blood- and lymph-channels in the intestine, lymph-glands, and many other organs. The newly produced cells may accumulate at the points of their formation or they may be carried to distant places. Endothelium is capable of forming connective tissue, and of participating actively in forming cicatrices. It may assume certain embryonal properties in inflammation and assist in the formation of granulation tissue. It has been shown, however, that when masses of fibrin in fibrinous exudates are replaced by connective tissue, the organization always proceeds from the point of their anchorage, and that this is true in spite of the fact that such masses may have been completely covered over by newly produced endothelial cells from the edges; therefore Le Count concludes that there is ample room for further proof of the production of fibrous tissue by endothelial cells lining the blood-vessels. He considers endothelial cells, however, very important factors in inflammatory reaction.

Brunn,[2] having studied the subject in man and performed certain experiments on the lower animals, states that the cells covering serous membranes belong to the group of epithelial cells, as proved by the facts that all of them possess a continuous row of very fine hairs, and that they show a tendency in a continuous manner to cover free surfaces and to prevent them from growing together. They may form, if inclosed in fibrin, and otherwise finding favorable conditions of growth, cystic, tubular, and adenomatous spaces, preventing at such points a development of connective-tissue adhesions. They never form connective tissue. New endothelial cells arise exclusively through a division of previously existing cells, and never from fibroblasts.

[1] Jour. Am. Med. Assoc., Dec. 7, 1901, p. 1521.
[2] Ziegler's Beiträge, 1901, vol. xxx, p. 417.

The Origin of Amyloid Substance.—An important study upon this subject is published by Browicz.[1] This observer, in an extensive study of the liver, found that even during life there is at times a separation of the capillary wall from the adjacent liver-cells, and that in the space thus produced hemorrhagic extravasations may occur. The erythrocytes in the latter may be preserved, or may be hyaline and fuse together into homogeneous masses. Sometimes these masses give the amyloid reaction. These metamorphosed erythrocytes may also take part in fibrin-formation or in a process simulating it. The author likewise found amyloid substance in the liver-cells, but does not think that this was due to any degeneration. He considers it to have been owing to the fact that the liver-cells have the power of englobing erythrocytes, and that the amyloid substance found in them was nothing more than amyloid red corpuscles. It is well known that amyloid substance has, in its beginning, a close relation with the blood-vessels, especially the capillaries. In the liver, it is found not only in the space between the cell columns and the capillary walls, but also in the walls of the interlobular veins and arteries and in the lumen of the intraacinous capillaries. The morphology of amyloid corresponds very closely with that of the masses produced by the confluence of altered erythrocytes. It is not likely that in the circulating blood amyloid erythrocytes are found, in view of the vast mass of red blood-cells. Browicz's conclusions are that amyloidosis is an infiltration of the tissues with extravasated, metamorphosed erythrocytes, or an accumulation of these latter within the blood-capillaries, in consequence of the slowing of the current. This view explains the localization, morphology, and close relation of amyloid substance to the blood-vessels. With regard to hyaline substance, the author believes that it may also be of erythrocytic origin, but that it is not to be viewed as an antecedent of myloid.

The Rôle of Certain Nongranular and Granular Somatic Cells in Infection.—Harris[2] discusses the best methods of preserving, repairing, and staining tissues for the purpose of studying granular and nongranular cells in diseased tissue. He concludes that plasma cells are derived from lymphoid cells by an increase in their protoplasm and by the formation around their nuclei of small, basophilic masses. They reach old inflamed areas, where they are so plentiful, by means of the lymphatics; and on account of the damaged condition of the walls of the lymph-vessels, and the greater amount of pressure in the diseased parts, the cells do not pass through but accumulate in and around these areas. Their appearance indicates the beginning formation of cicatricial tissue; they are the real fibroblasts. Endothelial cells are reproduced from pre-existing cells of their kind. Eosinophilic cells possess no significance in connection with infections. Mast-cells, of which there are 4 varieties (Ehrlich's, lymphatic, blood, and muscle), have a tendency to disappear in acute inflammatory conditions. In chronic processes they increase considerably in number; this peculiarity is of especial importance in differentiating so-called inflammatory conditions from sarcoma. Ehrlich's cell may be derived from either the large phagocytes of Metchnikoff, or from cells that are nearly related to them. Those in the lymph-nodes and those in the blood originate from the large hyaline cells that are present to a limited extent in this fluid.

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[1] Verhandl. d. Akad. d. Wissensch. in Krakau, math.-naturw. Cl., April 24 and July 1, 1901; Przeglad lekarski, 1901, Nos. 43 and 44; and Centralbl. f. allg. Path. u. path. Anat., 1902, vol. XIII, No. 3.

[2] Jour. Am. Med. Assoc., Mar. 18, 1902, p. 633.

Concerning the Regeneration of Lymph-vessels in Pleural Thickenings.—Talke,[1] having made histologic examinations of peritoneal adhesions and pleuritic thickenings, to determine whether or not lymph-vessels are formed in them, was able to demonstrate new lymph-vessels in a number of different forms: Endothelial tubes with simple walls; large lymph-vessels having 2 coats; plexuses; lymph-spaces in the interstices; perivascular lymph-spaces and vessels. They originate most likely from pre-existing lymph-vessels and spaces.

Fibrous Myositis.—Biggs[2] reports a case of fibrous myositis of the outer side of the right thigh. It had its origin in an injury to this region. Within 4 months a hard, prominent painful swelling appeared, which enlarged very rapidly. The skin over it was adherent to the mass; there was no enlargement of the lymphatic glands. Sections of the muscles were removed for microscopic examination. The muscle was firmer and paler than normal, and seemed to contain an excess of fibrous tissue. Upon microscopic examination some of the muscle-fibers were found to be atrophied, others undergoing degeneration. Fibrous connective tissue was seen everywhere to take the place of the muscle-tissue.

The Nature of Internal Lesions in Death from Superficial Burns.—To aid in determining the cause of deaths in superficial burns, McCrae[3] has examined histologically lymph tissues from 13 cases. He concludes his description by emphasizing the following points: The entire pathologic picture presents great similarity to the conditions found in the diseases characterized by the presence of toxins of bacterial origin in the blood. Damage to the lymphatic tissue is a constant feature, but is not necessarily focal, some cases presenting only diffuse degeneration. The cases which live but a few hours after infliction seem more likely to present a focal condition than those which live a longer time, as the condition which he interprets as proliferation and phagocytosis is one which may rapidly disappear. The focal lesions are not a true necrosis, but rather a proliferation of the endothelial cells of the reticulum and the capillaries, and a phagocytosis by the leukocytes and endothelial cells, to which latter is due the fragmented, disintegrated appearance which suggests a true necrosis.

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[1] Ziegler's Beiträge, 1902, vol. xxxii, p. 106.
[2] Univ. of Penna. Med. Bull., Dec., 1901, p. 353.
[3] Amer. Med., Nov. 9, 1901, p. 735.

NERVOUS AND MENTAL DISEASES.

BY ARCHIBALD CHURCH, M.D.,

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SYMPTOMATOLOGY AND SYMPTOMATIC DISORDERS.

Myopsychoses.—Joffroy[1] calls attention to the association of many so-called myopathies with mental disorders. Sydenham's chorea is almost always attended by mental dulness, while Huntingdon's chorea presents irritability and progressive dementia. Paralysis agitans is accompanied by a mild degree of dementia or at least by a condition of pronounced apathy. The automatic movements and cataleptic attitudes of adolescent insanity are the outcome of profound stupor with mental confusion. Progressive muscular atrophy is often attended by defective intellectual development with mental symptoms suggesting imbecility. Pseudohypertrophic paralysis in children and the juvenile myopathies are commonly associated with feebleness or defect of mind. Joffroy proposes for such association of motor and mental symptoms the term myopsychopathy, or, shortly, myopsychy. [The mental condition in paralysis agitans is usually better than superficial appearance indicates. There is mental inertia but rarely any dementia.]

Neurosis and Appendicitis.—Schauman[2] believes there is a distinct connection between the nervous diathesis and appendicitis. Very frequently members of the same family are attacked by appendicitis. The writer has collected 14 cases in which this has occurred. In 75 cases only one member of the family was affected, but there was marked evidence of neurotic tendencies.

Functional or Organic Paralysis.—Thomas Buzzard[3] again makes an important contribution to this subject, in connection with which his name is so familiar. He says he does not attach great importance to the presence of the stigmas of hysteria as of much assistance in the differentiation of functional and organic paralysis, for the reasons that, in the first place, although some of these may be present in a case of functional paralysis, it is not absolutely necessary that any should be discovered. Secondly, because such stigmas may not infrequently be found as a complication in a patient suffering from organic disease of the nervous system. Nor, in his opinion, is the presence of a neurotic history in the patient's family of any great help. He calls attention to the fact that there are symptoms the presence of one of which may be conclusive as to

[1] Rev. Neurol., April 15, 1902.
[2] Finska Lakare Handlingar, 1901.
[3] Brit. Med. Jour., Nov. 1, 1902.

the existence of organic disease. Among these he mentions optic neuritis or atrophy, fixed pupil, hemianopsia, absence of knee-jerks and definitely pronounced changes in electric reaction of the muscles, especially the reaction of degeneration, "picked out" atrophy of the muscles, this atrophy falling upon individual muscles, characteristic bedsores, paralysis of the bladder with ammoniacal urine, paralysis exactly limited to the district of a single nerve or other plexus, the toe-sign of Babinski. On the other hand, there are probably no symptoms which unsupported establish a diagnosis of functional paralysis. At the same time, the most important means is an accurate knowledge of individual symptoms incident to organic disease, and of equal importance an intimate acquaintance with the symptom-complex or association of symptoms which points with positiveness to special forms of structural disease. The mode of onset of the paralysis is also of importance. Paralyses of a functional nature are liable to be preceded by physical or moral shock, and present an appreciable interval of hours or days, and in the traumatic cases the physical injury is often insignificant in comparison with the succeeding palsy, in which also proper anatomic association is often lacking.

Sleeping Sickness.—W. B. Warrington[1] reports the condition of the central nervous system in a case of African lethargy. He confirms the findings of Mott in the Manson cases (see YEAR-BOOK for 1902), and his conclusions agree with those of Mackenzie. The etiology of the disorder still remains unknown, but the poison, of whatever origin or character, acts especially upon the lymphatic system, and particularly the lymphatic system of the central nervous apparatus.

Narcolepsy.—H. Stern[2] contributes an article on the subject of narcolepsy and other forms of sleepiness. He details a case which led to his investigation, and, in spite of his preconceived idea, was unable by the utmost care to detect any source of autointoxication. He is disposed to believe that a lack of chlorids is of importance in the production of the symptoms.

Acroparesthesia.—J. Collins[3] gives an analysis of 100 cases of acroparesthesia. The average age was 39, ranging from 17 to 79. Sixty-nine were females. There were 34 Americans, 41 Russians, and the rest were English, Irish, Scotch, German, and French. The onset was sudden in 14 cases out of 20 in which the pain was recorded. No attributable cause could be assigned in the great majority of cases. Among the alleged causes were noted rheumatism, la grippe, pregnancy, change of life, menorrhagia, constipation, fright, miscarriage, blood-poisoning, free use of alcohol, and intemperate use of tea. In half the cases the paresthesia was confined to the hands or arms; in 10 % to the feet or legs; and in 30 % it occurred in the upper and lower extremities. Only in one case was the face affected. The symptoms were described as numbness, staggering, pain, tingling, stiffness, creepiness, crawling, itching, burning, cold, wooden feeling. The most common association was some disturbance of the intestinal tract. Constipation 32 %, marked intestinal fermentation 10 %, anorexia and coated tongue 15 %. Of the cases 17 % complained of impaired dexterity. The average duration was about 14 months, ranging from 1 week to 6 years. The most important element in the treatment was attention to the intestinal tract. Prolonged rest, nourishing diet, new environment, are the requirements for a speedy cure. The paresthesia can be temporarily relieved by bromids, for instance, and other sedatives, but their use is undesirable. The application of electricity has been attended with gratifying results, both the galvanic and static currents being used in the cervical and brachial regions.

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[1] Brit. Med. Jour., Sept. 27, 1902.

[2] Med. Rec., Jan. 11, 1902.

[3] Med. Rec., May 31, 1902.

Tremor.—A very valuable and interesting paper on the subject of **the vibrations in ankle clonus** and tremor is contributed by J. A. McWilliam.[1] He recalls that a tendency to fusion in the vibrations of tremor and clonus had already been pointed out by Williams and Wolfenden in paralysis agitans and disseminated sclerosis particularly, detected by recording instruments. In paralysis agitans they obtained large curves at the rate of 5.1 a second, which were notched at the summit indicating a real rhythm of 10.2 a second. So in disseminated sclerosis, 5.2 large waves a second showed a true rate of 10.4 a second. The author noted that in ankle clonus a tracing obtained by recording instruments showed this double rhythm, and in one instance the rate of 14 a second was recorded. This is in contradiction to the statements of Horsley and Schaefer that the automatic action of nerve-cells of the cord appears to be capable of originating a rhythm of no greater frequency than 10 a second, and the discrepancy apparently is due to the fact that in ankle clonus only alternate beats are seen, two being fused and only discerned by the use of special recording appliances.

Family Periodic Paralysis.—J. K. Mitchell, S. Flexner, and D. L. Edsall[2] furnish a brief report of the **physiologic and chemical state** of 3 cases. The cases consisted of a mother and daughter, and a young man, who was one of 5 presenting the disease in 3 generations. The effect of the injection of blood-serum from patient No. 1 was tried in rabbits, the blood being taken during paralytic attacks, without presenting any conclusive results, but serving to confirm the usual negative findings regarding the toxicity of the blood. Examinations to determine the alkalinity of the blood during the attacks and during the interval by Löwy's method were made, but these investigations were unreliable, owing to the fact that the patient had taken large doses of alkalies. More important negative results were obtained from an estimation of the ammonia in the urine. Examination of the stomach-contents on two occasions gave a gross result showing that even starch digestion was not proceeding, and that there was total anacidity, the whole digestive process being absolutely at a standstill and gastric motor power diminished or abolished.

Oddo and Audibert[3] report cases of family periodic paralysis and embody a careful investigation of the literature of the subject. They have been able to find **64 published cases,** from which it appears that the disease develops between the fifth and thirty-sixth years of life. Most cases appear from the tenth to the twentieth year, and somewhat more frequently in the male than the female sex. In some cases, and this may be a family trait, the attacks appear after fatigue, in others during rest or even during sleep. The exciting causes of the attacks of paralysis are mild excitement, exposure to cold, excesses in food, and venery. Paresthesias, like feelings of heat and heaviness, are common in the affected parts, which finally present a relaxed motor palsy. They consider the impossibility of raising the head as tolerably characteristic, due to the involvement of the sternocleidomastoid muscles at the same time that the splenii remain more or less intact, so that movement of the head from side to side is maintained. They state that the face and eye muscles are never affected, but must have overlooked an American case in which involvement of the face was noted. In all cases studied by them the paralysis was symmetric, commencing in the legs, as a rule, and receding in the reverse order. Sensibility objectively is intact, while the electric reaction is reduced. The reflexes are lost during the attack, but reappear as the attack recedes. The sphincters are unaffected and the mental condition is unchanged, although occasionally somnolence is observed during the attack. The heart is sometimes affected, showing motor insufficiency, arrhythmia and dilation. The appetite is commonly lost, and sometimes a feeling of thirst is quite intense. The duration of the attacks varies from 15 minutes to 6 or 7 days, but is commonly from 2 or 3 to 48 hours. The intervals between the attacks vary from a few days to several years, and the appearance of the patients and the reactions to extraordinary tests in the intervals may be perfectly normal. These authors believe that the morbid process is muscular in location; in other words, that it is, as they say, a "myoplegia." As to its etiology, they have nothing definite to advance. Therapeutically they suggest the value of gymnastics and faradization.

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[1] Brit. Med. Jour., Nov. 30, 1901

[2] Brain, 1902.

[3] Arch. gen. de méd., Jan.-May, 1902.

Familial Nervous Diseases.—J. Baumlin,[1] in a very elaborate article on familial diseases of the nervous system, quotes a number of interesting cases showing a tendency of all the typical diseases of a familial character to drift into each other. One interesting case of Friedreich's disease subsequently presented disturbance of sensibility, atrophy, and pseudohypertrophy.

Alcohol and the Nervous System.—H. Friedenwald[2] briefly reports a few experiments upon the various alcohols on the ganglion cells of the retina. Different varieties of alcohol in small doses were exhibited in animals for a period of nearly 4 months. The animals were apparently all in good health when killed. The effects of 4 varieties of alcohol were alike, the retinal ganglion cells showed marked degeneration while the inner and outer nuclear layers were less affected. The action of methyl and ethyl alcohol was identical.

The Heel Tendon-jerk.—E. Bramwell[3] presents a study of the clinical significance of the absence of the heel-tendon jerk based upon the investigation of the sign. The following are his conclusions: (1) The Achillis-jerk is most easily and conveniently obtained in the kneeling position. The examination in the prone position is more difficult and requires considerable experience, although the results obtained in this position are almost equally reliable in the hand of a skilled observer. (2) The Achillis-jerk is (probably) constantly present in "health" in persons under 50 years of age. In other words the loss of the Achillis-jerk in a person under 50 years of age (in the absence of any local cause, such as great edema of the legs, which may mechanically interfere with the examination) is always to be looked upon as a sign of organic disease, with the same assurance as loss of the knee-jerk is so regarded at the present time. (3) In persons over 50 years of age the activity of the Achillis-jerk diminishes with increasing age, consequently in old people its absence, especially if bilateral, cannot be regarded as having the same diagnostic significance as it has in children, young persons, and middle-aged adults. (4) In the majority of the cases of disease in which the Achillis-jerks are absent the knee-jerks are also lost; *e. g.*, most cases of tabes, peripheral neuritis, etc. (5) In certain conditions in which the knee-jerks are unaltered the Achillis-jerks are found to be absent or greatly diminished. Under these circumstances loss of the Achillis-jerk may prove a valuable adjunct to diagnosis. (6) Since in tabes the Achillis-jerk is usually lost before the knee-jerk, its condition should always be ascertained in suspected cases of that disease (Babinski), and more particularly in those cases in which knee-jerks are present. (7) It has been shown by Babinski and others that in cases of sciatica the Achillis-jerk is usually lost on the affected side; the loss of the Achillis-jerk proves the existence of a sciatic neuritis. The absence of the Achillis-jerk is for this reason a physical sign of importance in the diagnosis between true sciatica and the hysteric form of the disease. (8) The loss of the Achillis-jerk caused by a sciatic neuritis may persist for years after the sciatica has itself disappeared. (9) Cases of multiple neuritis are met with in which the Achillis-jerks are absent, while the activity of the knee-jerks remains unimpaired. In such cases the absence of the Achillis-jerk is of value in diagnosis. (10) It appears probable that the Achillis-jerk is sometimes lost as a result of syphilis, just as the Argyll-Robertson pupil sometimes occurs as a result of that disease unaccompanied by other signs of tabes or general paralysis. (11) The absence of the Achillis-jerk is sometimes of value in localizing lesions in the spinal cord or the nerve-roots.

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[1] Deut. Zeit. f. Nervenheilk., Oct., 1901.

[2] Johns Hopkins Hosp. Bull., Feb.-Mar., 1902.

[3] Brain, 1901.

Perforating Ulcer.—Peraire[1] reports 2 cases of perforating ulcer of the foot completely cured by Chipault's method of stretching the plantar nerve.

Typhoid Spine.—E. G. Cutler[2] calls attention to typhoid spine as originally described by Gibney, and reports a case. He believes with others that the condition is due to periosteitis secondary to typhoid. In the same number of this journal F. T. Lord presents an analysis of 26 cases of typhoid spine culled from the literature. Of these 26 cases 22 were males. The only causative factor was typhoid, though mechanical strain is mentioned as a precipitating cause in 6. Pain was the constantly initial symptom, and the onset, as indicated by this symptom, varied from the latter part of the typhoid fever to 3 months after defervescence. Involuntary urination and defecation were present in 1 case; the knee-jerks were disturbed in 8, five times being associated with radiating pain. Symptoms of hysteria or neurasthenia were noted in 6 cases. Kyphosis or other deviation was definitely stated as absent in 8 and described as present in 8 cases. It generally involved several of the vertebræ. In general the pathology of the process seems to be a perispondylitis or spondylitis, or both. A few of such cases may be purely nervous. The coexistence of nervous symptoms in half of the cases suggests, however, that all the neurotic cases may be organic in fact.

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[1] Travaux de neurol. chirurg., 1901, No. 1.
[2] Boston M. and S. Jour., June 26, 1902.

Kernig's Sign in Brain Lesions.—Sailer[1] reports a case in which Kernig's sign was present on one side connected with a lesion of the opposite side of the brain in a laborer who presented left-sided paralysis of the face with Kernig's and Babinski's signs present only on the left side. The necropsy showed a focal encephalitis secondary to malignant endocarditis with a patch of hemorrhagic softening in the right motor area. In a second case the left knee-jerk was exaggerated and both Kernig's and Babinski's signs were marked on the left side. The patient improved under increasing doses of iodid, and Kernig's sign gradually disappeared. The left arm was completely flaccid.

F. S. Clark[2] reports 3 cases of meningitis in which Kernig's sign was **persistently absent.**

Intermittent Limping.—G. L. Walton[3] contributes the term **angina cruris** for this condition, and the term certainly has advantages, as the pain is the most constant feature. He reports a number of cases, discusses the literature, and concludes (1) The concurrence of the paroxysmal pains of angina cruris with pulseless pedal arteries is too constant to be explained by coincidence, though it is true that pulseless arteries may be found without the pains, and, conversely, that such pains may appear with apparently normal arteries; (2) the painful paroxysms are probably of vascular origin, and result from vascular spasm coupled, perhaps, with increased blood-pressure acting on vessels already partially occluded, whether from local or general disease (aneurysm, syphilis), from senile changes (atheroma), or from congenital tendency to angiofibrosis; (3) recurring painful cramps of constant seat probably represent a modified form of angina cruris; (4) it is important to examine the dorsalis pedis and posterior tibial, as well as the radial and temporal arteries, in all cases in which it is desirable to estimate the bearing of the vascular condition upon disease in the central nervous system or elsewhere.

I. H. Levy[4] reports a case of intermittent limping **terminating in**

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[1] Am. Jour. Med. Sci., May, 1902.
[2] Am. Jour. Med. Sci., May, 1902.
[3] Boston M. and S. Jour., April 3, 1902.
[4] Phila. Med. Jour., April 12, 1902.

gangrene.—The patient experienced cramps shortly after beginning to walk, but these disappeared after a short rest. The posterior tibial and dorsal arteries of both feet were pulseless; there was, however, a redness and swelling and sharp attacks of pain, especially at night when the parts were at rest, with marked hyperesthesia and hyperalgesia benefited by cold, aggravated by a dependent position, furnishing a typical picture of erythromyalgia. The case, therefore, is a mixed one, suggesting Raynaud's disease, erythromelalgia, and the arterial mischief which produces intermittent limping and gangrene, necessitating the amputation of the leg above the knee. Sections from the posterior tibial, anterior tibial, and other nerves, and of the vessels of the leg and foot, were made. The nerves were apparently normal, the arterial walls were thickened, the intima quite markedly increased, and the media was also somewhat thickened.

Intermittent Painful Paralysis of the Arms.—J. Massaut[1] reports an interesting case of painful intermittent paralysis of the upper extremities analogous to the intermittent limp which affects the lower extremities. In his case in both arms no arterial pulse could be found and the painful paralysis was of intermittent recurrence following the continued use of the members. After a few moments' use of the extremities pain commenced in the biceps and radial group of the forearm muscles and then the anterior group of the forearm. In conditions of cold the pain was more readily induced. It finally reached a degree that rendered the arms useless, the muscular power rapidly decreasing. This patient was a married woman of 24 years whose mother had had 8 stillbirths. The patient herself had aborted once and also had a child of 3 years, in good health, neither the patient nor her child presenting any evidence of luetic infection.

Myasthenia Gravis.—Long and Wiki[2] report a case with necropsy. A man of 46 presented the usual symptoms of asthenic bulbar paralysis, or, better, myasthenia gravis, from which he died. Postmortem the nervous system was by no means normal, but it was impossible to draw any parallel between the symptoms observed and the lesions found. These were diffuse, and affected chiefly the interstitial tissue, the ganglion cells of the nuclei, medulla and spinal gray being normal except in the anterior horn of the cervical region and a few cells in the nucleus of the twelfth cranial nerve. There was a slight amount of meningitis in the lower portion of the cord, some vascular changes in the vessels of the cervical enlargement, some scattered patches of sclerosis in the lateral region, and a well-marked patch of sclerosis in the lateral columns of the cervical cord. On the ground that this condition is due to a toxic state the writer is disposed to attribute the original cause to a condition of the lungs whence perhaps systemic infection was derived.

S. Goldflam[3] reports a case in which there were **decided changes in the muscles and muscle-bundles.** The patient presented disease of the lungs, and it is suggested that the conditions in the muscles might be inflammatory in character and metastatic in origin. Serial sections of the brain showed it to be entirely normal, while the muscular lesions were almost universal in distribution.

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[1] Ann. de la Soc. med.-chir. d'Anvers, Mar.-April, 1901.
[2] Rev. Méd. de la Suisse Rom., July 30, 1901.
[3] Neurolog. Centralbl., Feb. 1, 1902.

W. R. Gowers[1] calls particular attention to the **peculiarity of expression** in patients affected with myasthenia gravis. This consists in what he calls the "nasal smile," or the "levator smile," consisting in the absence of the normal movement at the corner of the mouth. In these patients the furrow of the smile is entirely above the upper lip at the wing of the nose. Three cases are reported in detail. In regard to treatment the author states that the effect of hypodermic injections of strychnin seems to be distinctly good, in one case its effect being marked and constant.

DISEASES OF SPINAL MENINGES AND SPINAL NERVES.

Herpes Zoster.—J. McF. Winfield[2] reports a further number of cases of zoster which he believes to be dependent upon the presence of the plasmodium of malaria in the blood. He reports altogether 24 cases, of which 14 gave positive evidence of malaria as proved by the presence of the parasite in the blood. Fifteen had intercostal zoster, 2 had the eruption on the face, neck, and shoulder, and in 2 the gluteal region was affected; 2 presented herpes on the neck and occiput; 1 was of the ophthalmic variety, another of the face and neck, another the lumbar region, and another showed a lesion beginning in the neck and extending down the arm. One case was bilateral, one was recurrent. The literature adds but little to the observations of malaria in relation to zoster. The author gives support to the contention of Head that in zoster we have an infectious disease which causes an inflammation of the root ganglia with consequent irritation of the nerves running from them, and believes that this affection is sometimes malarial.

Max Joseph[3] contributes a very important **statistical report** on the subject of herpes zoster, which throughout is compared with that of J. A. Cantrell, published in the same journal in March, 1898. In a general way it parallels Cantrell's report, and is based upon the records of 163 cases occurring among 15,603 patients with skin diseases in the dispensary for skin and venereal diseases in Berlin, Germany. The author draws the following conclusions: (1) In frequency, herpes zoster causes 1 % of all skin diseases. (2) There appears to be no difference in the occurrence of herpes zoster in men and women. (3) Herpes zoster is a disease of youth (15 to 30 years), noted, according to my statistics, in two-thirds of the cases. Before school age the disease is very rare, during school it becomes more frequent; after 30 years its frequency diminishes, and in old age it is rare. (4) So far as regards the region of body affected, herpes zoster is in general more frequent the more nerves supply the region. There is one exception to this rule, both from my observations and those of Greenough—namely, an exceptionally large number of cases in the trigeminal region. This may be perhaps due to the fact that the region supplied by the trigeminus is more open to injury and disease than any other region. (5) Both sides of the body are apparently affected with equal frequency, bilateral herpes zoster being rare. (6) Zimmerleni says that herpes zoster occurs among physicians and nurses in epidemic form; no other occupation seems to predispose to the condition. The final settlement of this point can only be made by consulting industrial and hygienic statistics. (7) Epidemics of herpes zoster occur in the spring and fall. Sporadic cases occur with almost equal regularity throughout the entire year, though some increase is noted in the spring and autumn months.

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[1] Brit. Med. Jour., May 24, 1902.

[2] N. Y. Med. Jour., Aug. 2, 1902.

[3] Phila. Med. Jour., Oct. 25 1902.

Meningomyelitis.—T. Diller[1] reports a case of meningomyelitis occurring during convalescence from typhoid fever. He refers to 3 cases reported by Curschmann terminating fatally. His own case also terminated fatally, and the microscopic and a careful bacteriologic examination of the spinal cord disclosed invasion by the typhoid bacilli, which could be demonstrated in transverse sections and in cultures. The patient had presented paraplegia, paralysis of the bladder and bowels, contractures of the legs, bedsores, intense hyperesthesia, limited to the portion of the body below the waist, exaggerated knee-jerks, making a diagnosis of meningomyelitis evident, but these symptoms appeared subsequent to the typhoid crisis.

Paralysis of the Peroneal Nerve.—Donath[2] reports the case of a primipara of 30 years who after narcosis and the extraction of the fetus had suffered pain in the right leg and also less marked pain in the left leg. Three weeks later the right leg was nearly entirely paralyzed, presenting the reaction of degeneration in the distribution of the peroneal nerve. Similar conditions in the left leg were less marked. Nadler[3] reports a similar case following a difficult labor, and both attribute the nerve-injury to conditions arising within the pelvis.

Sciatica.—F. Mendel[4] reports 3 cases of **syphilitic sciatica,** all relieved by mercurial treatment, and insists that in certain doubtful cases the use of mercury may clear up the diagnosis and at the same time cure the patient. In one instance treatment by the mouth and by inunction was without effect, but the sciatica disappeared rapidly after intramuscular injection.

Section of the Phrenic Nerve.—Schroeder and Green[5] contribute a very valuable article on the subject of the phrenic nerve. Section of the phrenic nerve in man has been supposed to be practically a fatal accident. This position is combated not only from experimental evidence, but by actual section of the phrenic nerve in man made necessary in the removal of a cervical tumor. The article, which is one of unusual worth, contains the following conclusions: (1) From the clinical and experimental data it would seem that the diaphragm is not an essential muscle of respiration; (2) that as the symptoms commonly described as caused by an irritation on the phrenic were uniformly absent not only in the operation, but in all of the experimental work as well, it is safe to infer that they may have been due to something other than a simple injury to the phrenic; (3) that while from an anatomic point of view the diaphragm undoubtedly is innervated by branches from the intercostal nerves, this nerve-supply is secondary to the phrenic and is insufficient to carry on the action of the diaphragm after a division of the phrenic. (4) that a division of the phrenic nerve, producing a partial collapse of the lower lobe of the lung on the affected side and an atrophy of one-half of the diaphragm, might predispose to infection of the lung or be followed by a diaphragmatic hernia; (5) that a division of one phrenic nerve in man, resulting in paralysis of one-half the diaphragm only, is not necessarily fatal.

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[1] Amer. Med., June 7, 1902.
[2] Pester med-chir. Presse, 1900, No. 50
[3] Correspondenzbl. f. Schweizer Aerzte, 1901, No. 18.
[4] Münch. med. Woch., July 2, 1901.
[5] Am. Jour. Med. Sci., 1902.

Paralysis of the Phrenic Nerve after Diphtheria.—W. McKenzie[1] reports 2 cases. The first was a boy of 6, who presented a general post-diphtherial multiple neuritis. Seventeen days after the paralsyis was first noted, and following a dose of medicine, the face became congested, the expression anxious, the temperature fell, there was dyspnea, the respirations were catchy, embarrassed, and shallow, the pulse was increased to 100, of poor volume and tension, and there was coldness of the extremities. The clinical picture suggested that of pulmonary embolism. The speech became monosyllabic and whispered. The abdomen, which before had been full and freely moving in respiration, was slightly hollowed and moved in a jerky spasmodic way. The condition increased, the abdomen became more retracted, the pulse more rapid, and finally death occurred apparently from heart failure. In the second case the patient was suffering from an ulcer of the foot, which was supposed to be syphilitic. He was treated with mercury and fomentations. The ulcer had almost healed when suddenly in the night a similar crisis occurred as in the preceding case and the clinical features were identical. Death occurred suddenly on the fifth day. A definite history of diphtheria was not obtainable in the second case, although there was a history of inflammation of the throat some 3 weeks before. In both cases the important clinical features were: (1) Enlarged thorax as compared to the abdomen; (2) abdominal excavation; (3) absence of abdominal movement beyond a jerky motion seen at the upper part; (4) monosyllabic whispering voice; (5) comparatively good pulse; (6) vomiting at the outset but not afterward; (7) reluctance to take food owing to the effort required; (8) fixity of the eyeballs at the onset; (9) development of pneumonia before death from heart failure.

Neuralgia of the Posterior Roots.—Chipault and Le Fur[2] report the case of a man of 48 who had an intense neuralgia about the trunk attended by an area of hypersensitiveness corresponding to the seventh, eighth, and ninth dorsal nerves and marked by crises of intense suffering resembling those in locomotor ataxia, but no evidence of tabes, other than the neuralgia and crises, was to be detected. An operation was done to divide the posterior roots of the involved nerves. The roots on the right side corresponding to the pain were embedded at their passage through the meninges in a layer of inflammation which was chronic and almost cicatricial in character. The whole of this was removed as well as 4 or 5 cm. of the eighth and ninth roots and a portion of the tenth. The dura was sutured and the wound closed. Inconvenience was caused by a considerable flow of spinal fluid and some hemorrhage, and the evening of the operation intense pain in the sciatic region was experienced, affecting first the left then the right leg, and attributed to a clot which had extended down from the point of operation as far as the roots of the sciatic nerve. It lasted 5 days. After the operation the patient experienced some relief from the previous unbearable crises, and anesthesia corresponding to the area of the roots removed was present. The writers consider the case as of importance as demonstrating that a localized meningitis causing intense pain from involvement of roots is open to surgical interference with reasonable promise of success.

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[1] Intercol. Med. Jour., Nov. 20, 1901.

[2] Gaz. des Hôp., Mar. 20, 1902.

Healing of Nerves.—Ballance and Stewart, in book form, Macmillan & Co., 1901, record experiments in the histologic investigation of the subject of healing of nerves. Of the two theories which are maintained to explain the return of function after division of a nerve, the authors' observations clearly support the "peripheral" as opposed to the "central" doctrine. The peripheral theory is that one according to which the new fibers in the distal segment are formed from pre-existing cells in the distal segment itself. Being thus laid down, they become attached later on to those of the central segment, thereby restoring the conductivity of the nerve-trunk. The authors therefore adhere to and amplify the results already obtained by Tizzoni, Kennedy, Bethe, and others. As regards the changes which characterize regeneration of a divided nerve, the authors' close observations show that they differ according to whether or not the proximal and distal segments have been brought into apposition. Even in the distal segment of a nonunited nerve regeneration of axis-cylinders and of medullary sheaths takes place, although full maturity of the new nerve-fibers is not attained unless the distal segment be joined to the proximal, so that their fibers may become functionally continuous. The earliest signs of regeneration occur at the end of 3 weeks. At this time may be seen spider-like neuroblasts shooting out young beaded axis-cylinders from their opposite poles. The new axons rapidly increase in length, and at the end of 4 weeks have grown so as to overlap and anastomose. The new medullary sheath makes its appearance about the fourth week, it too being laid down by a process of secretion along the side of a spindle-shaped neurilemma cell. Neither axis-cylinders nor medullary sheaths attain to full maturity unless the distal segment is joined to the proximal. Some stimulus evidently is needed to permit of their full development. They point out the importance of their findings in relation to the neuron conception, and are disposed to discredit the individuality of neuron elements, at least in so far as pertains to the peripheral nervous system. Here, they say, the nervous apparatus must be considered as made up of chains of cells set end to end, the axis-cylinder processes fusing together to form continuous paths.

Multiple Neuritis.—C. W. Burr and D. J. McCarthy[1] report a case of acute multiple neuritis with peculiar **changes in the gasserian ganglion.** In a summary they add a case of acute alcoholic multiple neuritis with **involvement of the bladder and rectum** and widespread degeneration of the central and peripheral nervous system involving the pelvic nerves, the vagus, and phrenic with hemorrhagic extravasation within the sheaths of the latter. Degeneration of both fiber and cellular structure of the gasserian ganglion was noted. The other intervertebral and cerebral nerve-ganglia were unfortunately not examined. It is demonstrated by this case that a multiple neuritis with demonstrable lesions affecting the bladder and rectal nerves can occur, and that too much stress must not be laid upon the usual escape of the sphincters in this disease in differentiating it from lesions of the cord.

F. Raymond[2] gives a résumé of the cases of multiple neuritis associated with **double facial paralysis.** He reviews cases of Althaus, Bregmann, Pugliesi, Preobrajenski, Brasch, and one of his own. He thinks that involvement of the facial muscles is very far from being so rare as is generally believed.

Guinon[3] reports the case of a child of 5 who presented unquestioned evidence of multiple neuritis following a typical attack of whooping-cough. This sequel has also been previously mentioned by Leroux and Moebius.

Ronald Ross[4] calls attention to the possibility of **beriberi** being in many instances, if not always, due to arsenical poisoning. He has previously published a case hailing from Sierra Leone in which the hair of the patient was found to contain arsenic. Certain other observers have noticed a similarity between beriberi and epidemic forms of neuritis due to arsenic, as, for instance, in the Manchester beer-poisoning. He has recently examined the hair from the heads of 20 patients suffering from beriberi, and in 6 instances found arsenic present; and a further investigation revealed the fact that in 5 out of the 6 cases presenting the arsenic the samples were taken during the first 20 days of the disease.

R. Ross[5] reports further instances of the presence of **arsenic in the hair of early cases of beriberi.** In 8 cases presenting a duration of from 3 to 73 days a trace of arsenic was found in 3 instances but none in the cases that had lasted over 30 days. In the same journal P. Manson, in opening a discussion on the subject of beriberi, suggests the following basis upon which to found a recognition of the disease as a distinct form of multiple peripheral neuritis: (1) Our ignorance of its cause; (2) its occurrence as an endemic and epidemic disease; (3) its proneness to produce cardiac disability and dropsy; (4) nonimplication of the cranial nerves, except the pneumogastric; (5) nonimplication of the intellectual and emotional centers; (6) rarity or complete absence of trophic skin lesions; (7) high rate of mortality under certain conditions. He believes that the immediate cause of beriberi is a toxin, and that this is produced by a living germ which is produced by some culture-medium located outside the human body; that this toxin does not necessarily enter the human body in food or drinking-water, but is associated with the surroundings of the patient.

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[1] Phila. Med. Jour., Nov. 2, 1901.
[2] Lectures on Diseases of the Nervous Systèm, Paris. 1901.
[3] Jour. de Méd. et de Chir. Prat., Oct. 10, 1901.
[4] Brit. Med. Jour., Feb. 8, 1902.
[5] Brit. Med. Jour., Sept. 20, 1902.

DISEASES OF THE SPINAL CORD.

Family Spastic Spinal Paralysis.—W. G. Spiller[1] records the history of a family presenting spastic spinal paralysis. He reports and illustrates 2 cases of father and son, and the family history shows that the affection has run through 8 generations. The disease usually appears before or about the fifth year of life and presents uniform symptoms. It has been more common in the female sex. The elder of the two cases reported was a man of 32 years, a telegraph operator. The disorder is said to have begun at the age of 5. At the present time the limbs are not wasted. The left foot is in equinovarus position, the right foot less contractured. The left foot is shorter than the right, the instep very high. The patient has little voluntary movement of the toes or feet, the weakness being marked on the left side. Knee-jerks very much exaggerated, ankle clonus and Babinski sign on each side. The feet are sensitive in walking, the gait very spastic. The muscles respond to the faradic current, but a strong one is required. The patient has the powerful grasp of the hand suitable for his occupation.

Primary Spastic Paraplegia.—Erb,[2] in a lecture before the Post-Graduate College of the West London Hospital, gave particular expression to his views on the subject of primary spastic paraplegia, which he was one of the first to establish as a morbid entity. As far back as 1875 he described cases presenting the 3 cardinal symptoms of feebleness in the lower extremities, muscular rigidity in the same region, and increased reflexes. Years passed before pathologic anatomy was able to confirm the presumptive deductions then established. In this lecture he was able to give the details of 10 cases examined by various observers showing sclerosis confined mainly to the lateral tracts and principally to the pyramidal bundles. He insists upon the autonomy of the disorder, particularly as it is found in family groups, which have been observed all over the world. The only difficulty, as far as diagnosis is concerned, in a case which is open to continuous observation, is presented by multiple sclerosis, and here the addition of symptoms on the part of the upper levels, the intention tremor, nystagmus, and difficulties of speech, serve to differentiate very clearly when they have once developed. In the same lecture Erb reviewed the subject of syphilitic spastic spinal paralysis and reiterated his contention that it furnishes at least a clinical type, if not a nosologic entity, consisting in the involvement of the pyramidal tracts mainly and the cord secondarily, and as not being secondary to meningitis, myelitis, or other local accident of syphilitic or other origin. He insists that in a sense it is primary as far as location is concerned, though induced by syphilotoxins, in the same sense that tabes is a local disease of the posterior tracts due to similar causes.

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[1] Phila. Med. Jour., June 21, 1902. [2] Brit. Med. Jour., Oct. 11, 1902.

Lesions of the Conus Medullaris and Cauda Equina.—B. W. Sippy,[1] an author who has given this subject very careful study, finds by charting a number of cases that the work of Starr relative to the posterior thighs is well confirmed and the charts of Head, Kocher, and Wichmann are not accurate. He believes, however, that further study will lead to modifications of all existing charts. The differential diagnosis, the point of major importance and one sometimes impossible of determination, particularly engages his attention. Except when due to trauma, disease of the cauda, he says, usually develops slowly, producing symptoms more or less characteristic of root disease.

Spinal Paralysis in Syphilitic Infants.—Peters[2] calls attention to paralysis of the arms and legs from changes in the spinal cord due to infantile syphilis. The most striking feature of the paralysis is the abnormal position of the hand and forearm, called the "flipper position," due to a paralysis of the radial nerve and muscles and the strong action of the pronators. It usually occurs the third or fourth month of life, coming on rather suddenly. It is noticed that the child cannot move one or both arms, and sometimes the legs are also involved. The affected limb is limp and hyperesthetic, but sometimes the shoulder muscles alone suffer, or the whole arm may be powerless except some power in the flexors and pronators. It is probable that cases formerly classed as syphilitic pseudoparalysis are of this nature. They were attributed to arthritis preventing the child from using the limb, and it was thought that no real paralysis existed. The tenderness is attributed by Peters to the involvement of the sensory as well as the motor root in the cord. If the upper portion of the cord be affected, the pupil is often implicated through its sympathetic fibers, presenting a contracted condition with sunken eyelids. Sometimes the knee-jerks are lost when the lumbar cord is involved, and the lower limbs are then correspondingly affected. The disease was noted in 11 instances, and in all cases receded promptly upon the use of mercury and iodid, consequently no necropsies have been had, yet Peters is convinced that the lesion is in the cord.

Brown-Séquard's Paralysis.—A. R. Edwards[3] reports in full detail a case of this clinical syndrome, and properly points out that conclusions based upon experiments on animals are not applicable in this connection to the conditions found in the human being.

Transverse Lesions of the Cord.—W. A. Turner[4] carefully reviews the subject of transverse lesions of the cord in relation to the knee-jerks, and reaches the following conclusions: (1) The condition of the knee-jerks after experimental transection of the spinal cord in monkeys is not constant: the higher the level of the transection, the greater the likelihood of the knee-jerks being temporarily diminished or abolished. In these observations there is found a confirmation of the experimental data given

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[1] Jour. Am. Med. Assoc., May 10, 1902. [2] Jahrb. f. Kinderheilk., Mar., 1901.
[3] Jour. Am. Med. Assoc., Mar. 15, 1902.
[4] Jour. Nerv. and Ment. Dis., June, 1902.

by Rosenthal and Mendelsohn, and by Gad and Flatau in dogs. (2) No such difference according to level is found in transections of the human spinal cord; all complete transections above the lumbar enlargement lead to abolition of the knee-jerks, most probably of a permanent character. But temporary abolition of the knee-jerks, sometimes for a prolonged period, may follow incomplete anatomic transection, although the other coexisting phenomena point to a complete physiologic lesion. (3) In man, as experimentally in monkeys, although the knee-jerks may be abolished, some true reflex actions are permanently maintained; such are the plantar and the superficial anal reflexes; and, notably in monkeys, the crossed adductor jerks. (4) Accepting the view that the state of the knee-jerk indicates the degree of the neuromuscular tone, if some other muscular action dependent upon "tone" is found to be abolished in spinal transection as well as the knee-jerk, it may legitimately be argued that neuromuscular tonus is impaired or abolished in physiologic transection; such other atonic paralysis is, I submit, to be found in the "yawning" of the anus, which has been described. (5) Therefore, we may conclude that in spinal transection in man, and in high transections in monkeys, actions dependent upon neuromuscular tone are permanently or temporarily abolished; but that true reflex movements are not impaired. (6) The variation which exists in the phenomena following lesions at different spinal levels in dogs and monkeys, the transitory effects of such lesions as regards the knee-jerks, as well as the temporary abolition of the knee-jerks in incomplete lesions in man, would preclude the general application of Bastian's theory; for there is nothing yet recorded to negative the view that the mechanism which produces loss of the knee-jerks in man, or their temporary abolition in the lower animals, is not to be found in the spinal cord itself. (7) The explanation of the discrepancy which exists between the results of transection in laboratory animals and man may be explained by the greater autonomy of the spinal segments in maintaining neuromuscular tonus as we descend the vertebral scale.

Hereditary Cerebellar Ataxia.—H. T. Patrick[1] reports a case of hereditary cerebellar ataxia and discusses the bearings of this subject generally. The article is one of unusual value and merit, and should be read in full. The author does not believe that the time has yet arrived in which sharp distinctions can be made among the various clinical types of hereditary ataxia. He does not think that Marie's cerebellar variety can be sharply differentiated from Friedreich's cases, and finds intermediate instances of which the Friedreich type and the cerebellar type are the extremes. Very careful investigation of the literature has been made, and the bibliography appended to the article is one of value.

Spinal Fracture.—G. L. Walton[2] presents a contribution on the study of spinal fracture with special reference to the question of operation. He concludes: (1) There are no symptoms which establish (otherwise than through their persistence) irremediable crush of the cord.

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[1] Jour. Nerv. and Ment. Dis., Mar., 1902.
[2] Jour. Nerv. and Ment. Dis., Jan., 1902.

(2) While total relaxed paralysis, anesthesia of abrupt demarcation, total loss of reflexes, retention, priapism, and tympanites, if persistent, point to complete and incurable transverse lesion, the onset of such symptoms does not preclude a certain degree of restoration of function. (3) The prognosis without operation is grave. (4) While the results of operation are not brilliant, they are sufficiently encouraging to warrant us in making the practice more general. (5) In most cases it will be wise to operate within a few days of the injury, but a delay of some hours is advisable, partly on account of shock and partly to eliminate the diagnosis of simple distortion. (6) We have no infallible guide to the extent of the lesion. The operation, at the worst, does not materially endanger life nor affect unfavorably the course of the case, and may at least reveal the lesion and lessen the pain; it may sometimes save a patient from death or from helpless invalidism of most distressing character. Instead of selecting the occasional case for operation, we should rather select the occasional case in which it is contraindicated (the patient with great displacement of vertebræ, the patient with high and rising temperature, the patient plainly moribund, the patient still under profound shock). (7) The dura should be opened freely; it need not be sutured; drainage is not necessary.

Suture of the Spinal Cord.—F. T. Stewart and R. H. Harte report a most interesting and suggestive case in which the spinal cord was entirely severed and the ends brought together by operation with partial return of function. The patient was a waitress, 26 years of age, admitted to the Pennsylvania Hospital January 21, 1901. She had received a bullet wound from a 32-caliber revolver. At the time of the accident the patient experienced the impact of the bullet, then a mighty contraction of the lower extremities, followed by a sensation as if the lower part of the body had been lost. Consciousness was not disturbed. The bullet entered about an inch to the right of the seventh dorsal spine, entering the spinal canal. There was immediately a complete abolition of motion and sensation below a line transecting the lower part of the tenth dorsal spine, at a point about 3 inches below the umbilicus. The line of demarcation was sharply defined and was superimposed by a belt of hyperesthesia reaching as high as the ensiform cartilage. Just before operation this hyperesthesia became anesthetic. By operation the bullet and a number of small fragments of bone were found between the ends of the severed spinal cord opposite the seventh dorsal vertebra. The fragments of bone and bullet were removed with the lacerated nervous tissue, leaving a distance of ¾ inch between the segments of cord. This condition was observed by a number who were present. The ends of the cord were approximated with chromicized catgut passed through the entire thickness of the cord transversely. The twenty-first day after the operation the patient felt urine in the bladder but could not void it. She could feel pin-pricks as far as the umbilicus and behind as far as the sacrum, but could not localize them On the sixtieth day the patient was in a chair, able to move the toe readily and with effort could flex the knees. In the eighth month the patient used the bed-pan and urinal and was able to stand by supporting herself between the bed and a chair. The ninth month she could feel the floor under her feet and appreciate the position of the feet; controlled her bowels, except when the feces were liquid, and rarely soiled herself with urine. The fourteenth month the patient in a tub-bath, can feel the water on her lower extremities and distinguish between that from the hot and the cold spigots. Sixteenth month the bowels move every other day and are under control except when diarrheic. About a pint of urine passes every 24 hours; sometimes there is incontinence in sleep. The patient has the senses of touch, pain, and temperature all over, although the difference between heat and cold is not always satisfactorily detected. A pin-prick can be differentiated from several pin-pricks, and pin-pricks from a sharp blow with a pencil, as low down as the knee, but the localization of this sensation is not accurate. The muscles are rigid, and there is marked but easily exhausted ankle and patellar clonus. The deep reflexes are marked and can be reinforced by muscular exertion of the face and arms and by painful sensations. On tickling the sole of the foot the big toe flexes. No bedsores have ever developed. [It is to be hoped that opportunity may furnish parallel and confirmatory cases, as the results in this instance are so much at variance with experience as to raise serious doubts and hopes.]

Muscular Atrophy.—P. Marie[1] illustrates some of the peculiarities of body formation in the so-called primitive myopathies. Two brothers show the flattening of the occipital region, previously noted in a communication by Marie and Omanoff in 1891. In these cases, owing to the wasting of the sternomastoid, the neck is given an appearance of additional width and flatness with prominence of the bony chain constituted by the superior borders of the clavicles and the sternum.

F. Allard[2] reports a case that in contour and history conforms to the myopathic type, but presents the **peculiarity of retaining galvanic and electric response** even in the muscles which are notably atrophied.

C. E. Beevor[3] reports a case of the **family type of congenital spinal muscular atrophy.** This case was a child who was noticed at birth to be paralyzed in all its limbs and all the trunk muscles with the exception of the diaphragm and the neck muscles, so that all the muscles except the diaphragm and the face muscles and those supplied by the cranial nerves were involved. All the muscles were much wasted, and not one gave reaction of degeneration to the constant current. There was analgesia and probably also anesthesia of the whole trunk and of the limbs. The determination of this was difficult in a child so young, but marked signs of analgesia were nevertheless present at all levels below the second rib. Plantar reflex present, knee-jerk absent, cremasteric well marked, vesical reflex performed naturally, tonus of the sphincter ani maintained. Out of 8 children, 3 others were affected like the present case. Although they were apparently born healthy, at the end of a month or six weeks they became weak, in one case the arms preceding the legs, and the muscles wasting. The change in the cells of the cord was that which is found in progressive muscular atrophy of spinal type. They were small and had lost their cell processes and possessed very little chromophilic substance. There was no engorgement or cell infiltration that would signify a myelitis. As the cells were diminished in number, it seems probable that many of them had disappeared, and it is possible that some of them had never been formed. Loss of sensation is attributable to degeneration in the posterior columns of the cord, which was found. Degenerate fibers were found in both brachial plexuses, especially on the right side, possibly due to traction on the arms at birth.

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[1] Nouvelle Icon. de la Salpêt., Jan., 1902.

[2] Nouvelle Icon. de la Salpêt., April, 1902.

[3] Brain, 1902.

Hereditary Cerebellar Ataxia.—Switalski[1] reports a case of hereditary cerebellar ataxia with postmortem findings. The patient was a man 41 years of age. The disease had been observed for 8 years previous to his death; he had been under the care of Charcot and Londe. On section the cord and cerebellum showed diminished proportions throughout. In the cord there was degeneration of the columns of Goll, the direct cerebellar tract, Gowers's tracts, and atrophy of the gray substance without disappearance of many cells from the anterior horns. In the bulbar region degeneration of the direct cerebellar tracts and of the tracts of Goll. There was also considerable atrophy of the direct cerebellar peduncle with proliferation of connective tissue below the ependyma of the fourth ventricle and in the aqueduct of Sylvius. The cerebellum showed a diminution in a number of its convolutions, which were separated by very deep fissures and a general reduction of white substance. The right optic nerve was atrophied; in the peripheral nerves and in the roots of the spinal nerves the large fibers had apparently disapeared with an augmentation of the number of small fibers. These observations correspond fairly well with those of others now on record, especially the one reported by Mendal.

Tumor of the Spinal Cord.—W. K. Hunter[2] reports a case of tubercular tumor of the spinal cord in a child 2 years old. A swelling in the cervical portion of the cord occupied 5 segments or about 1½ inches, beginning at the second pair and ending at the seventh. On section it was found to be a tuberculous nodule, oval in shape, about ¾ inch in length and occupying the center of the cord, with a narrow rim of tissue surrounding it. [The age of this patient is the earliest, as far as can be recalled, of the appearance of a tumor of the spinal cord.]

M. A. Starr[3] reports a case of removal of **fibroma in the upper dorsal region of the spinal cord.** The diagnosis was clear, and guided by the prominent symptom of pain, but the operation was delayed to try antisyphilitic treatment, which the author considers a mistake, as thereby irreparable damage to the cord was permitted through the further growth of the tumor. The tumor was found 2 inches higher than was anticipated, and the author calls attention to the necessity of going upward always when the tumor is not found at the point selected and indicated by the ordinary charts. In this case the tumor lay 6 inches above the line of anesthesia and 8 inches above the level of the pain.

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[1] Nouvelle Icon. de la Salpêt., Sept., 1901.

[2] Brain, 1902.

[3] Phila. Med. Jour., Feb. 28, 1902.

H. Oppenheim[1] details a case of **spinal cord tumor** located opposite the arch of the eighth dorsal vertebra, at which point the upper limit of the growth was exposed. It was subdural, of a pale red color, somewhat firmly attached to the arachnoid, but shelled out without disturbing the cord, which was pushed to one side by the pressure. The growth was situated to the right and behind the cord. Its longest diameter was 3 cm. It was about the size of a date. It proved to be a fibroma. The patient made a good recovery, the symptoms of paraplegia disappeared, the gait became quite natural with no trace of spastic paresis 2 months after the operation. Curvature, which had been present previous to the operation, also became markedly less.

Myelitis.—Winscheid[2] has reported the case of a woman who has apparently had two attacks of myelitis **in puerperal periods.** He observed the patient in her second confinement, when she presented paralytic symptoms 5 weeks after a normal birth. The illness came on with violent vomiting lasting several days, followed by paralysis of both legs, which set in rapidly, and there was great difficulty of urination. Two weeks later the paralysis was complete, the patellar reflexes greatly increased on both sides, and sensation abolished almost to the umbilicus. The muscles responded to electricity. A month later sensation in all the muscles was restored and the motor paralysis was confined to one side. Skin reflexes were absent and the patellar reflex and ankle clonus less marked. Complete incontinence of the urine had existed for some time, but now the urine could be held and controlled. There was no fever throughout the illness. The previous puerperium had been attended by exactly the same conditions, as described by the patient. The etiology of the condition is not explainable. There was no evidence of disease of the vertebral column.

H. D. Singer[3] concludes that **thrombosis of the spinal vessels** is the most common cause of the symptom-complex of acute myelitis, and that it is the consideration of greatest value. He believes that prompt treatment in cases presenting premonitory symptoms and a history of syphilis may ward off an attack of this nature; and to avoid confusion he believes we should follow Bastian and classify these cases as thrombotic softening of the cord, or as spinal thrombosis.

Spinal Cord Lesions Due to the Severe Anemias.—This subject has received a number of contributions, among which may be mentioned the Shattuck lecture delivered in Boston by Frank Billings, of Chicago.[4] Besides presenting the literature of the subject, Billings gives a detailed history of a large number of cases and presents the microscopic specimens derived from several. Archibald Church[5] gives the clinical characteristics of these cases of pernicious or very severe anemias attended by spinal cord changes. He lays particular stress upon the fact that the symptoms are not always those of a flaccid paralysis, but, on the contrary a spastic condition may be associated with this state, with increased reflexes, and in some instances flaccidity and spasticity may alternate a number of times. Paralysis of the bladder and bedsores may attend the spinal involvement. The further fact brought out by his cases, and definitely stated, is the tendency of the disease to assume an ascending form, and, in given instances, the body-levels are gradually but progressively involved from below upward, furnishing a clinical type which is suggestive of a Landry paralysis, though of much more tardy progress. In the way of treatment he has found that the intestinal injections of large quantities of normal salt solution have a beneficial influence on the general condition of the patient, without, in his experience, modifying the blood state aside from increasing its volume.

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[1] Berl. klin. Woch., Sept. 20, 1902.

[2] Centralbl. f. Gynäk., 1902.

[3] Brain, 1902.

[4] Boston M. and S. Jour., Sept., 1902.

[5] N. Y. Med. Jour., July 26, 1902.

Landry's Paralysis.—T. Diller[1] furnishes a study of Landry's paralysis with a report of 3 nonfatal cases. From general considerations he suggests that the term Landry's paralysis be employed, as neurologists, for instance, employ the term apoplexy In other words, that it be considered as generic, embracing cases some of which can be made out to be cases of poliomyelitis, others of multiple neuritis, as well as those which can be grouped under either head and present negative postmortem findings.

Poliomyelitis.—Frank R. Fry[2] reports an interesting case of acute anterior poliomyelitis in a youth of 18 in which **decided sensory disturbances** were present. After considering this particular feature of poliomyelitis, the author lists the following possibilities: (1) One may encounter cases with no sensory symptoms. These are rare in adults and in children old enough to express themselves. (2) Cases are encountered with severe pain and hyperesthesia in the affected part at the onset. These cases are also rare. (3) Cases are observed in which sensory disturbances present a mild degree of severity. These are common. Further, the author believes that cases are encountered in which multiple neuritis and poliomyelitis are associated. Such cases have, indeed, been pathologically demonstrated. They are difficult of diagnosis, but fortunately not frequent, and seem to be caused by widespread and intense toxic processes producing grave general symptoms.

D. H. MacKenzie[3] reports 10 cases of poliomyelitis occurring in **epidemic form,** to which he adds 7 others occurring in the same locality not under observation by himself. There were, all told, 7 deaths. Out of the 17 cases only 2 presented the classic symptoms of poliomyelitis. One was undoubtedly cerebral spinal meningitis. Several presented symptoms of acute ascending myelitis of Landry's type, and several showed involvement of cranial nerves. Some of the cases also presented symptoms singificant of peripheral neuritis; that is to say, there was more disturbance of sensation in the extremities than is supposed to attend a poliomyelitis. [These cases show the usual heterogenous clinical picture presented by epidemic poliomyelitis. Some cases, if occurring sporadically, would be considered instances of polioencephalitis, others as instances of ascending myelitis, still others as cases of myelitis. It is apparent that one and the same infection is capable of producing a large variety of clinical pictures dependent upon the manner and location of its invasion.]

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[1] Jour. Ment. and Nerv. Dis., Oct., 1902.

[2] Jour. Am. Med. Assoc., May 19, 1902.

[3] Med. Rec., Oct. 4, 1902.

E. W. Taylor[1] produces a number of general considerations under the title of **poliomyelitis of the adult,** and suggests the following conclusions as a practical clinical guide and as a working basis for further study: That adult poliomyelitis is a well-marked clinical affection, characterized by initial fever, rapid onset of usually extensive paralysis, motor in type, with a tendency toward recovery, though often resulting fatally, from respiratory paralysis. That the disease has frequently been confused with multiple neuritis and so-called Landry's paralysis. That its anatomic basis is a primary inflammation in the distribution of the ventral arteries of the cord, leading to a destruction of nerve-cells. That this inflammation is rarely sharply limited to the ventral horns, but extends into the dorsal gray matter, the surrounding white matter, and at times into the oblongata. That there is no sharp line to be drawn between these lesions and still more extensive ones giving rise to totally different clinical pictures; *e. g.*, encephalitis, polienecephalitis, polioencephalomyelitis. That, therefore, anatomically the disease is much less sharply characterized than it is clinically. That its final place must be determined by a study of its cause or causes, as related to various other degenerations and inflammations of the nervous system. That from the practical point of view it is well to consider those cases poliomyelitis which show a flaccid atrophic paralysis of sudden onset, with definite anatomic changes limited to the ventral horns of the cord and their immediate vicinity.

Tabes Dorsalis.—B. Bramwell[2] furnishes an **analysis of 155 cases** of tabes of which his knowledge is complete. Regarding etiology he finds 3 factors: (1) The organic constitution of the nerve-tissues of the individual and the vulnerability of the nervous tissues in each case; (2) syphilis, the action of which he regards more as a predisposing than as an exciting or direct cause; (3) nervous and cord strain or irritation due to excesses from exertion, irritation by toxins, etc. In his analysis of individual symptoms he finds that in 148 cases in which **lightning pains** were present, they were in every case felt in the legs, whether they occurred in other parts of the body or not. In large portions of these cases the pains were said to be worse during bad weather, and in many cases worse after the patient got warm in bed. In several cases where optic atrophy developed the pains lessened with the loss of sight, but this was by no means invariable. In 114 cases in which **anesthesia in the leg** was reported upon, it was present 68 times and absent 46 times. In many of the cases in which it was present it was slight in degree. Anesthesia of the face was specifically mentioned 58 times as being absent, and was present only in 2 cases. **Ulnar analgesia** was present in 29 out of 60 cases. The **ataxic gait** was present in 120 of the 155 cases.

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[1] Jour. Ment. and Nerv. Dis., Aug., 1902.

[2] Brain, 1902.

The author found that rubber heels and soles could be used by many of his patients with benefit in improving the gait. **Incoordination of the arms** was present in 39 out of 108 cases. **Incoordination of the legs** was present in 117 out of 152 cases. **Hypotonia** of the muscles was present in 38 out of 47 cases in which it was investigated. The **knee-jerks** were ultimately lost in 132 of the 155 cases. In 2 cases in which the knee-jerks were absent when the patient was first seen they returned as the case progressed, and eventually were again lost. The **Achilles-jerk** was examined in 52 cases and was found absent 44 times. In 7 of 8 cases in which the Achilles-jerk was present on both sides the knee-jerk was also present on both sides. Investigation for **Babinski's sign** in 47 cases resulted as follows: In 3, no movement of the toes; in 39, flexion of the toes; in 5, extension of the toes on one or both sides. The author states that in only 2 cases of tabes has he seen the Babinski sign present in which he could not satisfactorily account for it by some associated lesion or complication. In 115 out of 144 cases the **bladder reflex** was affected in some way or another. In 99 cases the **sexual reflex** was affected 73 times, unaffected 26 times. The association of the cremasteric reflex in this connection shows that in those cases of tabes in which the cremasteric reflex is affected the sexual reflex is much more frequently affected than unaffected. In 104 cases in which the **size of the pupils** is specifically noted they were found contracted 69 times, medium size 27 times, dilated 8 times. Light reflex was lost or sluggish in 121 of the 155 cases. In 24 cases the pupils reacted promptly to light, but in 10 cases the condition of light reflex was not noted specifically. Argyll-Robertson pupil was present 90 times on both sides and in 5 cases on one side. In 13 cases an incomplete Argyll-Robertson pupil was present on one or both sides. In 37 cases in which the light reflex was affected and in which syphilis was denied the Argyll-Robertson pupil was present in complete form in 22 and in incomplete form in 7. In 4 cases the pupils contracted neither to light nor to accommodation. **Optic atrophy** was present in 33 out of 55 cases, or 60 %, a somewhat larger proportion than has been generally given. The **pulse** was examined in 25 consecutive hospital cases and the average of frequency estimated for the first 12 days after admission. In 9 the average pulse frequency was above 85, a percentage which corresponds with the reports of Riley. Out of 155 cases, 12 developed **general paresis** and probably 4 others. This, coupled with the fact that in 75 of the cases the disease is still in progress, makes it probable that general paresis develops in a percentage considerably larger than 10.

Disturbance of Sensation in Tabes.—F. Raymond[1] has reported a number of cases of tabes in which the **cutaneous and deep sensation** has been carefully studied and is graphically demonstrated. He finds that the disorders of sensation in locomotor ataxia present as particularly characteristic a polymorphism and dissociation and geometric segmental distribution. The dissociation which is so familiar in cases of syringomyelia may be typically shown in locomotor ataxia as well as in insular sclerosis and syphilitic myelitis, and as a general rule in these affections it depends upon alterations of the central gray matter of the cord. In syringomyelia the author points out that by contrast tactile sensibility is commonly respected, that the limits of disordered sensation are decidedly defined and are practically the same for analgesia and for thermoalgesia, while in tabes all phases of dissociated sensibility are the rule, extending by overlapping and irregular areas. The author believes that the study of the objective disorders of sensation in tabetics furnishes a further argument for recognizing that the essential seat of the tabetic process is not in the roots of the cord but in the cord itself.

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[1] Lectures on Diseases of the Nervous System, Paris, 1901.

J. H. W. Rhein[1] calls particular attention to the **involuntary movements** present in locomotor ataxia. These are much more common than is usually supposed and are worthy of serious attention. He divides such involuntary movements into: (1) The associated movements, which may exist also in combined systemic disease of the spinal cord, cerebral hemiplegia, spastic spinal paralysis, and cases of unilateral lesion of the spinal cord; (2) sudden twitchings of the trunk or extremities, which may occur with or without pain, and are more frequent at night; (3) twitchings in isolated muscles or parts of a muscle; (4) fibrillary twitchings; (5) rhythmic tremor resembilng that of paralysis agitans; (6) passive movements, which Hirschberg describes as being those movements of the legs or thighs which occur when a patient who is lying down attempts to sit up in bed without the assistance of his hand; or when he coughs; (7) involuntary movements previously described as athetoid movements.

A very rare association of organic lesions, **tabes dorsalis and multiple sclerosis,** was shown in a case presented by Wharton Sinkler before the American Neurologic Association.[2] The patient was a man of 62 who, besides presenting the ordinary signs of tabes, also presented a distinct intention tremor, which is shown in his handwriting or attempt to use his hands for any purpose whatever, and in the usual tests. The speech and eye symptoms were not present, although the patient's age leads to the possibility of the tremor being partially senile in character, although it is so thoroughly developed and so pure that it can only with difficulty be attributed to the man's age.

The treatment of locomotor ataxia by the **exercise method** of Fränkel is receiving favorable comment from all sources. Strozewski,[3] Panski,[4] and Navarro[5] all report cases greatly benefited and continuously improved by this method.

G. M. Holmes[6] gives a very concise and practical outline on the subject of the **etiology of tabes.** The main interest will be found in his exposition of the views of Edinger's compensation theory. This is based upon the well-known physiologic fact that function implies consumption of part of the active tissue. Edinger has applied this view to the nervous system, and assumes that if the supply of nutrition be deficient, repair is disturbed by overactivity. To the success of his theory it is essential that it be proved that certain tracts of the nervous system under normal conditions are working more than others, all being subjected to the same noxious influences. He contends that the sensory tracts are in constant activity, while the motor tracts are only temporarily or momentarily put into operation; that consequently under these conditions the lack of muscular tone and deep reflexes would naturally be expected to occur first, thereby inducing ataxia; that the anesthesias and retarded sensibilities would naturally follow, and through the constant modification of the iris by efforts at accommodation and variations in light, pupillary symptoms would be readily induced; thus, the Argyll-Robertson pupil, ptosis, ocular paralysis, and optic atrophy might theoretically be expected to occur. In the same way the bladder function, which is in constant operation, would more readily yield than some others. In studying that hypothesis Edinger has sought to establish his ideas by experiment after the manner of Voss, who has produced extremely depraved nutritional conditions in animals by the use of substances which destroyed their blood-making powers; such animals, then being subjected to forced activities, have presented degenerations closely resembling those of tabes in the motor tracts of the cord. It will be observed that there is nothing in Edinger's theory which controverts the importance of the specific element in the etiology of tabes, but rather serves to explain why it is that the morbid condition, which is general, should present pathologic changes which are limited. The involvement of the lower extremities is due, according to Edinger, to the fact that the lower extremities, as far as fatigue is concerned, are much more frequently used than the upper extremities. In some cases of tabes, particularly in the upper end of the cord primarily, he has found that the subjects were those who made great use of the arms, as artisans, seamstresses, and in one case a policeman seemed at first to controvert the theory, but it was found that he was a mounted officer and used his legs very little, but constantly employed his arms in the use of the reins. The progressive character of tabes, pauses and sudden increase and pathologic processes, are also explained from this standpoint. Edinger insists that exertion and fatigue of any sort should be avoided, and maintains that in a large number of cases treated for a number of years on this basis rather satisfactory results have been obtained in the way of retarding the progressive development of the disease. In the same way he explains the well-known clinical fact that when optic atrophy appears ataxia is likely to subside, on the ground that blind men do not overexercise, and functional wear and tear is consequently at a minimum. The great proportion of males over females is attributed to similar causes. The limitation of the pathologic process to the intramedullary portion of the sensory neurons is explained as follows: (1) The peripheral nerves are provided with an additional sheath which may be an additional protection, and (2) the least resistant point of the neuron is at its point of entrance into the cord.

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[1] Jour. Nerv. and Ment. Dis., July, 1902.
[2] Phila. Med. Jour., Oct. 25, 1902.
[3] Medycyna, 1900, No. 10.
[4] Czasopismo lekarski, 1901, No. 12.
[5] Travaux de neurol. chir., 1901.
[6] Dublin Jour. Med. Sci. Nov., 1901.

Pansini[1] reports 2 cases of tabes apparently **due to lead-poisoning.**

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[1] Riv. Crit. di Clin. Med., Jan., 1902.

The author calls attention to the painlessness of forced extension of the knee, and thinks this may be an early premonitory sign. [The question of a syphilitic infection in these cases is not adequately answered.]

W. B. Cornell[1] reports an interesting case of locomotor ataxia with **osteoarthropathy of the vertebral column** demonstrated clinically and confirmed by radiography. He refers to the 14 cases collected by Abadie, this case making 15 in all. Scoliosis was the most common curvature: the dorsolumbar variety 7 times, cervicodorsal 4 times, dorsal 3 times, lumbosacral once and multiple scoliosis once. Kyphosis was the next in frequency, 3 times in the dorsal region, twice in the dorsolumbar region, twice in the cervicodorsal region. Lordosis occurred 3 times. A knuckle was present in 5 cases. The pathology is that described in tabetic arthropathies in general, the hypertrophic variety, however, being the most frequent.

DISEASES OF CEREBRAL MENINGES AND CRANIAL NERVES.

Meningitis.—E. Blavot[2] says that the treatment of meningitis with **hot baths,** recommended by Aufrecht in 1894, combined with lumbar puncture, has proved almost invariably successful in cases presenting various degrees of severity. In grave cases the prolonged bath of 25 minutes, renewed every 3 or 4 hours day and night, was used with success. The patient may be removed to and from the bath in a blanket. Infants and grown-up patients stand the treatment equally well. Lumbar puncture he believes to be a powerful adjuvant, as it diminishes the intracranial pressure and the toxic substances. Nine patients from 2 to 14 years of age were treated. The author believes the prognosis is good when the continued treatment by hot baths and lumbar puncture is used. The baths should be continued until convalescence is fully established; they are sedative and promote sweating and diuresis. Lumbar puncture should not be followed by aspiration of the fluid. It may be done daily until the fluid is normal in appearance microscopically. In addition, saline transfusion may be practised twice daily, calomel and opiates given internally, and an ointment having iodoform as its base applied to the head and neck.

Jassniger[3] reports a case of purulent cerebrospinal meningitis presenting **Friedländer's pneumococcus** in pure culture in the lesion. The infection was traced from the nose to the sphenoidal sinus, which was filled with thick pus, thence through the walls to the cavernous sinus. Here a phlebitis started which had spread to the whole of the cerebrospinal system.

The **character of the cerebrospinal fluid in meningitis** has received considerable attention from Widal, Sicard, and others, and their contention has recently been supported by Milian, who asserts that the discovery of uninuclear leukocytes in meningitis points to the tuberculous type, whereas polynuclear cells indicate an acute inflammation. Against this conclusion Marconi-Mutzner[1] warns the observer. He relates the history of a case that died with symptoms of basal meningitis; the fluid obtained by lumbar puncture contained polynuclear leukocytes exclusively, but at the autopsy a tuberculous condition was demonstrated. He also disagrees with the opinion that lumbar puncture is a harmless procedure.

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[1] Johns Hopkins Hosp. Bull., Oct., 1902.
[2] Thèse de Paris, 1902.
[3] Centralbl. f. Bakt., July 12, 1901

Pachymeningitis Hæmorrhagica Interna.—J. O. W. Barratt[2] contributes an important paper on this subject. He believes that intravascular separation of fibrin is an apparently constant event in subdural membrane formation. Such subdural membranes are free from micro-organisms as far as revealed by staining methods. The results of culture upon agar indicate that during life such membranes are free from bacteria. He found that experimental implantation of false membrane in the subdural space of cats failed to set up a progressive process of membrane formation. The changes which occur are comparable to a process of repair. He advances the view that pacyhmeningitis hæmorrhagica interna is a morbid process in which the essential lesion is a separation of fibrin, usually within the blood-vessels, upon which the other conditions of change are largely, if not chiefly, dependent. John C. Munro[3] calls attention to the clinical symptoms in pachymeningitis and the value of operating to remove the clots. This has been done in several instances with good results. He concludes: (1) Hemorrhagic pachymeningitis is found in the insane and in infants, but for the most part in men not insane after middle life. (2) Alcohol, syphilis, acute and wasting diseases, and trauma apparently bear some causative relation. (3) The symptoms are those of diffuse subdural hemorrhage, coming on slowly, producing mental irritation, spasm and rigidity of the extremities, convulsions, and, later, paralysis, the sequence being more or less irregular. (4) The cranial nerves are not liable to be affected. (5) The treatment is surgical, and should be instituted as early in the disease as possible. (6) Without operative relief in cases with pressure symptoms the prognosis is practically hopeless. Early operation is not serious and gives the best chance for recovery.

Trifacial Neuralgia.—Spiller and Frazier[4] report upon the proposition of **dividing the sensory root** of the trifacial nerve, instead of removing the ganglion in the cure of obstinate trifacial neuralgia. They conducted a number of experiments in which the proposed operation was practised on dogs, and Spiller, from the neurologic side, reached the conclusion that regeneration of the fibers with restoration of function does not follow division of the sensory root. The operation proposed is practically the Hartley-Krause procedure for the removal of the ganglion, but the purpose is to spare this organ and divide the sensory root of the fifth before it enters the ganglion. The operation has been put into effect in one instance by Frazier—a man of 68 who suffered from trifacial neuralgia for 5 years, for which neurectomy of the supraorbital nerve had been performed 4 years previously, followed within a year by a second peripheral operation to meet the requirements of the disorder, with relief for 6 months, then a third and a fourth operation, affording relief for progressively shorter periods of time. October 12, 1901, operation was done, dividing the sensory root of Gasser's ganglion. November 2d the patient was discharged from the hospital, having had no recurrence of pain. At that time examination revealed absence of the supraorbital reflex on the affected side and anesthesia of the entire trifacial distribution. In this operation the motor root had also been divided. [This operation as described and with the accompanying remarks is doubtless less difficult than the removal of the ganglion, but still is attended with decided technical obstacles, and must be of limited value, only to be successfully carried out in the hands of the most skilful. It would seem possible to open the skull and reach the nerve within the dura by raising the lower surface of the brain, as has been done by Horsley, and so come upon the nerve without the bloody dissection the Hartley-Krause route entails.]

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[1] Arch. Gen de Méd., 1901.

[2] Brain, 1902.

[3] Chicago Med. Recorder, Dec., 1902.

[4] Phila. Med. Jour., Dec. 14, 1901.

Frazier and Spiller[1] **report further upon the case of trifacial neuralgia** upon which they operated about a year ago by division of the sensory root of the ganglion. At the time of the first report only about 2 months had elapsed; now, at the end of a year, the relief from pain has been absolute and the condition of sensation remains the same, but through division of the motor branch there is permanent paralysis of the muscles of mastication on the affected side, constituting an annoyance but no great difficulty. In a foot-note to the same article Spiller remarks that W. W. Keen has also exsected the roots of the gasserian ganglion in another case, and was not able, nor were those present at the time, to recognize the motor root from the sensory. Keen expressed the opinion that from an operative point of view this procedure was very much less difficult than excision of the ganglion. Spiller contends that after division of the sensory root, and probably much of the motor root, regeneration does not take place, from the fact, as pointed out by Bethe and Ballance and confirmed by Stewart, that the presence of the neurilemma sheath is essential to regeneration in nerves, contrary to the ordinary teaching. The absence of the neurilemma sheath on the roots of the fifth nerve above the point at which it enters the pons therefore makes it subject to the same rules as pertain to other intracranial tracts.

Delageniere[2] reports 2 cases of facial neuralgia treated by **section of the cervical sympathetic,** at the same time by resection and forcible stretching of the branches of the fifth nerve distributed to the painful areas. In one case there was slight but temporary amelioration, and in the other no appreciable relief was obtained.

G. Cavazzani[3] reports 8 cases of trifacial neuralgia **said to be cured by resection of the superior cervical ganglia of the sympathetic,** relief being attributed to modification in the nutrition of the gasserian ganglion. In none of the cases, whether operation was limited to the ganglion or included a larger portion of the cervical chain, were there ill effects. The author thinks very highly of the procedure.

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[1] Phila. Med. Jour., Oct. 25, 1902.
[2] Travaux de Neurol. Chir., June, 1901.
[3] Gaz. degli Osped., Jan. 26, 1902.

P. F. Arullani,[1] on the ground that this neurosis is **analogous to epilepsy,** employed **Flechsig's treatment of epilepsy,** consisting of the administration of opium in gradually increasing doses for 2 months, then its abrupt discontinuance and the substitution of bromid of potassium for 2 months, also in increasing doses. He reports 4 out of 5 cases cured, and so remaining at periods varying from 18 months to 3 years. The fifth case relapsed and was cured by operation. The author believes this should be tried in all facial neuralgias before having recourse to operation. It is necessary that the patient should be kept in bed, carefully dieted on milk, and abstain from alcohol.

[The bed and milk régime was probably the most important element in the treatment.]

Facial Paralysis.—Breavoine[2] discusses the method of Faure and Furet of treating facial paralysis of traumatic origin by **establishing anastomosis** between the involved facial nerve and the spinal accessory. In 2 cases in which this operation has been performed it was successful in one, and in the other, regeneration of the nerve was established beyond doubt, although the result was not altogether satisfactory. He insists that the operation should not be attempted unless it is certain that the facial palsy is beyond all other methods of cure. The operation is difficult and delicate, but apparently free from risk, the resulting paralysis of the trapezius being extremely limited and without functional importance. In the operation both nerves are divided, the facial near its exit from the stylomastoid foramen, and the part of the spinal accessory which supplies the trapezius muscle just before it enters the sternomastoid, the central end of the latter being attached to the peripheral end of the facial.

H. O. Reik[3] reports cases to support the theory that in Bell's palsy the involvement of the facial nerve in the aqueduct is commonly due, in the so-called rheumatic instances, to an extension of the **catarrhal or other inflammatory process in the middle ear.** He reports 4 cases in some detail in which the tympanum was involved in a catarrhal process prior to the onset of the paralytic symptoms, and he believes that if cold is the cause of most cases of facial palsy, it acts through the otitis media, whereby the nerve is involved in the inflammatory process through its exposed sheath or pressure upon it by an exudate. He believes in paracentesis of the tympanic membrane, and urges that in every case of facial palsy the ear be examined. Anatomically he finds that in quite a large proportion of the cases the nerve is exposed to irritating infection by middle-ear processes lying superficially in the tympanic cavity.

DISEASES OF THE BRAIN PROPER.

Brain Tumor.—Charles K. Mills[4] reports a case of brain tumor localized clinically and by the **x-ray.** The tumor was found in part and enucleated by operation, and at the necropsy the remaining portion, which was very large, was found. In shape and location it conformed to the x-ray pictures. Some experiments were also made, at Mills's suggestion, by Pfahler, for the purpose of detecting intracranial conditions. Tumors of various sorts were inserted into the brain of a cadaver and in some instances corresponding shadows were obtained. The investigators conclude that: (1) Fibrosarcomas, and probably other tumors, can be photographed in the living subject and their location and extent shown; (2) various tumors can be photographed in their most common locations; (3) other abnormalities and deficiencies in brain-tissue itself can be photographed, which will probably be of value in the diagnosis of cysts, softening, and hemorrhages; (4) overexposure of the third series and underexposure of the fourth show that good results will follow only the most careful technic and keen judgment as to the special conditions in each case; (5) the shadows obtained in normal parts of the brains studied indicate that great care is necessary in the interpretation of any shadow obtained in the living subject. These conclusions are very much in keeping with those already presented by Church,[1] who was the first to detect a brain tumor by the x-ray in the living subject, and he is given due credit in the article in question. Mills's case is the second one of the sort to be reported.

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[1] Gaz. Med. di Torin., Sept. 10, 1901. [2] Travaux de Neurol. Chir., June, 1901.
[3] Johns Hopkins Hosp. Bull., April, 1902. [4] Phila. Med. Jour., Feb. 28, 1902.

Putnam and Williams[2] discuss the symptomatology of **tumors involving the corpus callosum.** They point out that symptoms arising from such tumors may be readily mistaken for psychoses of various sorts, and still more for the symptoms of general paresis. They state that perhaps the only symptoms attributable to the injury to the corpus callosum itself are the mental changes. In one instance which they report a point of special interest was the early appearance of slight changes in character, a slight impairment of mental power and memory, preceded by other symptoms of brain involvement. There were, however, isolated epileptic seizures several years before, and this has been noticed several times in cases previously reported. Even when the tumor attains large proportions and the destruction is apparently very great, damage to the brain mechanism is usually first revealed through disturbed mental action. The conclusions reached by various writers upon the subject of tumors located in the corpus callosum agree on general points: the usual pressure signs are long delayed, and of positive symptoms the mental disorder is of the greatest interest. This takes the form of mental failure, rapidly developing stupor, or of hallucinations, irritability, or attacks of excitement, etc., if the progress of the case is not very rapid. Paralysis, when present, is usually first hemiplegic and of the ordinary type, but tends to become diplegic, particularly as regards the legs, and this is highly characteristic. Convulsions, when they occur, are liable to be bilateral. On the other hand, the general pressure symptoms of brain tumor often fail to appear until a late period, and cranial nerve palsies are generally wanting. Tendon reflexes may be exaggerated or unchanged. A peculiar ataxia has been noted by some observers, a sort of lack of balance and a lack of motor initiative or poverty of movement. The authors tabulate a number of cases obtained from the literature and add their own, on which they base the general conclusions outlined above.

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[1] Am. Jour. Med. Sci., Feb., 1899.

[2] Jour. Nerv. and Ment. Dis., Dec., 1901.

M. F. Porter[1] reports an interesting case of brain tumor in which the **diagnosis was complicated by multiple neuritis.** The symptoms pointed to the right motor region, which was uncovered surgically. The sense of increased resistance was found with a needle and the finger, at a depth of about half an inch below the surface, and by means of the finger the tumor mass, ovoid in shape and of a little greater consistency than the brain, with a slight capsule allowing an easy separation from the surrounding brain-tissue, was removed. The tumor was about the size of a pullet's egg. Since the date of the operation symptoms indicate a recurrence of the growth.

E. E. Laslett[2] reports an interesting case of tumor of the pons associated with **degeneration in the posterior columns of the cord.** A peculiarity in the clinical history was the appearance of a peculiar fever presenting a curve suggesting a prolonged remittent type. The degeneration of the cord in this case was confined to the outer margin of the posterior external columns, and by the author is attributed to traction upon the first cervical posterior roots owing to pressure and displacement of the parts at the foramen magnum.

M. L. Perry[3] reports a case in which the **pineal gland** was involved in a tumor. The case indicates that a tumor of considerable size may involve the pineal gland without giving any pressure symptoms, and that the pineal gland may be entirely destroyed by disease without presenting any signs.

James Stewart[4] reports an interesting case of **cerebral glioma.** A man of 37 years suddenly became speechless for about 20 minutes. There were recurring similar attacks at short intervals for several weeks; then headache, optic neuritis, and facial spasm, followed by paralysis of the lower facial group of muscles with weakness of the right half of the tongue. There was marked dysarthria, first for the labials, and, later, for all letters and words, and finally complete speechlessness. On operation a tumor was found beneath the gray matter of the lower part of the ascending frontal convolution. It was firm and encapsuled and readily removed, followed at first by an increase in all symptoms and then by their disappearance. Two months afterward the patient was apparently perfectly well. The tumor upon investigation proved to be a glioma. The author lays stress upon the dysarthria as indicating, in accordance with the statements of Bastian, a subcortical location in this territory.

Burr and Taylor[5] report a very severe case of tumor of the brain relieved by operation, but followed by hemiplegia with **muscular atrophy** and death 10 months later. The case was noteworthy for its long duration —namely, 7 years. The Jacksonian epilepsy constituted the only evidence of brain disease for a long period of time. The other symptoms of new-growth appeared comparatively late. The occurrence of muscular wasting on the palsied side, after operation, is also extremely interesting. The author calls attention to the fact that true muscular atrophy after cerebral palsy is accounted for by several theories, and states that the weight of evidence is in favor of the opinion that the cells of the anterior horns are the true trophic centers of the muscles, particularly as the percentage of cases with evident spinal cord disease is constantly growing smaller under better methods, and he therefore concludes that the wasting in these cases must be due to the secondary disturbance of the spinal cord brought about by an unknown mechanism.

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[1] Jour. Am. Med. Assoc., Jan. 25, 1902.
[2] Brit. Med. Jour., Mar. 22, 1902.
[3] Med. Rec., Nov. 2, 1901.
[4] Amer. Med., Dec. 21, 1901.
[5] Am. Jour. Med. Sci., July, 1902.

Cerebellar Tumor.—G. von Voss[1] reports 5 cases of cerebellar tumor and gives an interesting table of their **symptoms.** Dizziness was noted in 4; vomiting in 3; pain in the head in 5; and ataxia or similar motor disturbance in 5; stiffness of the neck in 3; disturbance of the facialis in 4; disturbance of the eighth nerve in 4; one-sided atrophy of the tongue in 2; optic neuritis or atrophy in 5; dementia or stupidity in 5; and slow pulse in 4. A number of other symptoms were also tabulated, but these show the most important relations. The knee-jerks, for instance, were increased in 3 cases, normal in 1, absent in 1, and the muscular tone was increased in 3, reduced in 1, and normal in 1.

Piollet[2] reports 2 cases of **cerebellar surgery** under the care of Jaboulay; one for a tuberculous deposit in the right lobe, not treated by operation, the other for glioma, removed by trephining, followed by permanent improvement. He appends a table constituting, with these cases, 48 records of tumor of the cerebellum removed by trephining. The results from this table are such as lead to unfavorable conclusions regarding surgical measures in cerebellar tumor. In 20 cases death ensued. Improvement was noted in 16, but was followed by early recurrence, resulting in death. Almost complete cure followed 4 times, and rapid but seemingly complete cure in only a single instance.

Concussion of the Brain.—Hauser[3] relates a case of fatal concussion in a patient aged 56. The symptoms gradually increased in severity up to the sixth day, when death occurred. Blows in the right temporal region produced a fissured fracture extending to the base. No hemorrhages of any size, only some infiltration of hemorrhagic matter at the point of impact and the opposite point, were discovered. No signs of compression anywhere. Throughout an area corresponding to the line between trauma and contrecoup there were patches of commencing softening without hemorrhage. At these points granular degeneration was found microscopically, involving the white matter and the ganglion cells, and is believed to have occurred as a direct consequence of the concussion. [The clinical definition of concussion and contusion, etc., is a useless effort and an impossible task, as one condition shades into the other through all possible gradations. It is entirely a question of degree.]

The Lip and Tongue Centers.—C. L. Dana[4] reports a very interesting case bearing upon the localization of the lip and tongue centers, calling attention to the misleading conclusions that have been derived from experiments upon apes and animals. He concludes that in the human brain the lip and tongue centers are closely connected and more or less identical; that one group of centers for the tongue is in relation with the articulatory movements of the lips and another with the movements of mastication, of opening and closing the mouth, and of swallowing. He believes that the area for the excitation of movements of the tongue is a wide one, associated probably in its lower parts with movements of articulation and in the upper parts with chewing movements. These centers lie at the base of the anterior and posterior central convolutions.

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[1] Deut. Zeit. f. Nervenheilk., Dec., 1901.

[2] Arch. Provinc. de Chir., 1901.

[3] Deut. Arch. f. klin. Med., Bd. LXV.

[4] Phila. Med. Jour., Feb. 28, 1902.

Lesions of the Quadrate Bodies.—F. Raymond, in his fifth series of lectures on disease of the nervous system (Paris, 1901), apropos of a case in which there was a lesion in the region of the corpora quadrigemina, enumerates the symptoms of this condition as follows: (1) There is a diminution of vision which may reach complete blindness, accompanied or not by dilation of the pupils. It is more likely to accompany lesion of the anterior quadrigemina than the posterior pair. (2) Paralysis of the associated movements of the eyes, but principally of the lateral movements. These palsies are generally partial. (3) Pupillary disturbances, which may be unilateral. They consist in paralytic mydriasis and in the more or less complete abolition of the light reflexes and those of accommodation. (4) Disorders of sensibility in the form of paresthesia, and which in certain areas may reach the degree of complete anesthesia, often involving muscles and joints. A one-sided lesion of the quadrate bodies produces these sensory disorders upon the opposite side of the body, which are attributable to compression or invasion of the sensitive pathway. (5) Toxic disorders appear to depend in large part upon the disorders of sensibility, and especially of the deep-seated anesthesias. They consist commonly in awkwardness in executing intentional movements, sometimes in tremor provoked by the execution of voluntary movements; disorders of equilibration and in walking, recalling the cerebellar conditions, are also encountered. (6) Athetoid and choreiform movements are generally present. (7) Diminution of hearing on opposite side indicates that it is contralateral. It is to be regarded as an evidence of the invasion of the posterior quadrate bodies. (8) Difficulty of mastication is exceptional, but to be explained by the extension of the morbid process to the motor nucleus of the fifth nerve or to the root of this nerve.

Acute Hemorrhagic Encephalitis.—E. Straeussler[1] reports 2 cases of acute hemorrhagic encephalitis attributed to autointoxication arising from the intestinal tract. In the first case the intestines contained hard roundish fecal lumps with corresponding swelling and redness of the mucous membrane. In the rectum a circular fold of reddened and swollen mucous membrane occluded the bowel. In the second case the necropsy showed various sized patches of cortical softening in the brain with atrophy and chronic internal hydrocephalus. There were hemorrhages into the subdural spaces and in the cortex, various hemorrhages in the intestinal mucosa, and a fine pigmented flat scar in the ascending and transverse colon and two smaller ones in the descending and transverse colon. The large intestine was blocked with hardened feces.

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[1] Wien. klin. Woch., No. 3, 1902.

Abscess of the Brain.—R. W. McClintock[1] reports a case of abscess of the brain due to Bacillus typhosus, which was isolated and cultivated with all necessary care. The cerebral involvement followed a typical attack of typhoid.

Internal Hydrocephalus in the Adult.—F. P. Weber[2] makes a study of internal hydrocephalus in the adult, and reaches the following conclusions: (1) That the various kinds of hydrocephalus and effusions into the ventricles of the brain may be fitly compared to the various kinds of effusions into the pleura and the peritoneum. (2) That the cases of so-called idiopathic or simple internal hydrocephalus are probably nearly all due to more or less localized serious meningeal or ependymal inflammation, and are strictly analogous to cases of serous effusion into the pleura or peritoneum resulting from localized nonsuppurative pleuritis or peritonitis. It is of course probable that there are several different microbic or toxic agents. (3) That the reason why chronic inflammatory thickening of the membrane stretching from the cerebellum to the medulla and forming part of the roof of the fourth ventricle has to often been found present in fatal cases of chronic hydrocephalus is not necessarily merely that the foramen of Magendie and the neighboring foramina have been closed by inflammation (as they very often must be in similar cases), but that this portion of the roof of the fourth ventricle is one of the sites of election for the localized inflammation which leads to hydrocephalus. (4) That the theory of a purely angioneurotic effusion to account for some cases of acute internal hydrocephalus has as yet not sufficient evidence to support it, though doubtless the amount of irritation required to produce the same pressure of effusion varies according to conditions (temporary or persistent, congenital or acquired) of the blood-vessels and lymphatics which influence the local circulation; doubtless also temporary circumstances, such as sudden change from the upright to the recumbent position, and vice versa, mental or physical exertion, undue exposure to cold or heat, the ingestion of alcohol or other stimulants, and reflex effects on the local circulation from other parts of the body, may influence the tendency to effusion in the ventricles. (5) That many of the cases of apparently acute hydrocephalus in adults and older children are really exacerbations of a chronic condition, sometimes, doubtless, dating from early childhood or birth, as evidenced by the history of previous cerebral symptoms, by the relatively large size of the head, or by the postmortem evidence of association with a condition of hydromyelia or syringomyelia, or, as in the present case, by excessive weight of the brain.

Multiple Insular Sclerosis.—J. Hoffmann[3] gives a very admirable résumé of this subject. His remarks are based upon 100 cases, 43 women and 57 men, which have been under his own observation. In 5 % of these cases infectious disorders seem to play some causal part.

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[1] Am. Jour. Med. Sci., April, 1902.
[2] Brain, 1902.
[3] Deut. Zeit. f. Nervenheilk., Dec., 1901.

Syphilis was not accused in any instance and metallic poisoning was not noted, but one case had come in contact with lead in any way. Alcohol and sexual excesses played no part. A neuropathic tendency was not more commonly found than in other sorts of diseases. Certainly in 10 cases, and probably in 13, trauma seemed to have been the important factor, while shock and strain were not considered to have played a part. In over half of the cases no causal factor was discoverable. Under the symptomatology he refers to the polymorphous clinical character of the disorder. In 54 of his cases he found changes of articulation, such as monotonous voice, stumbling, muffled speech, slurring or scanning speech. Giddiness was present in 64 %, headache in 40 %. Commonly the temporal half of the optic nerve was found to be blanched and in many cases atrophied. In 50 % optic atrophy was present. The nystagmus was commonly horizontal in range and was present in 56 % of the cases. He considers nystagmus as simply an intention tremor of the eye muscles. In 71 % of his cases the intention tremor in the hands was decidedly marked. In regard to sensation, 66 complained of modifications of the sensation of feeling, and in 28 it was objectively determined. Commonly paresthesias of taste, pain and temperature conditions, and other common cutaneous parasthesias were mentioned by the patients. Objectively hyperesthesia or anesthesia for all qualities of the sensation of feeling, or dissociated sensations of feeling or disturbances of the stereognostic sense were obtained.

Paralytic Attacks.—A. Pick[1] furnishes a paper on the subject of the **treatment of paralytic attacks,** containing several important suggestions. He calls prominent attention to the danger menacing these patients by reason of infection through various portions of the alimentary canal and the necessity of seeing that intestinal disinfection is maintained. This means cleanliness of the mouth and its adjunct cavities; activity of the intestinal tract, and antisepsis as far as it can be reasonably carried out. He also calls attention to the necessity of watching the alimentation of the patient, and especially the necessity of seeing that they have fluid food which is properly introduced into the stomach, and that danger of lung infection by the introduction of fluids into the trachea be carefully avoided. He calls attention to the great necessity for supplying water to the system during the first few days, when the patient usually cannot swallow, the need of water being greater than that of food, and suggests the use of rectal enemas and hypodermoclysis.

Cerebral Hemianesthesia.—Dercum and Spiller[2] report a very interesting case showing that organic hemianesthesia may be caused by a lesion of the sensitive crossway and lenticular nucleus without involvement of the optic thalamus except that due to degeneration. They are not sure that the involvement of the lenticular nucleus has anything to do with the hemianesthesia. Almost the whole of the posterior limb of the internal capsule was affected, which seems to indicate that the sensory fibers are located chiefly, if not entirely, in the area of the sensory crossway, but it may be that some sensory fibers pass through the lenticular nucleus.

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[1] Phila. Med. Jour., Feb. 28, 1902. [2] Am. Jour. Med. Sci., Mar., 1902.

Pseudohypertrophy Following Infantile Cerebral Palsy.—B. P. Clark[1] discusses the subject of hypertrophy of the muscles—a comparatively rare condition—in infantile cerebral palsy. He believes that this increase in the muscular mass is due to the association of the peculiar cortical lesion with some degree of posthemiplegic disorder of motility in parts once paralyzed. In other words, that when there is a tendency to posthemiplegic overactivity or athetoid movements, pseudohypertrophy of the muscles may appear. Even bony hypertrophy has taken place in some cases, as proved by the author in one of his instances by x-ray examination.

Cerebral Hemorrhage and Softening in Infants.—Porak and Katz[2] report the case of an infant born spontaneously, somewhat asphyxiated but easily revived. It was fed artificially. On the fifth day the temperature fell to 95° F. and there was general icterus. Convulsions occurred on the eighth day. Four days later the left lower extremity was in contraction, the temperature fell to 93° F., and the weight fell from 5¾ to 4¾ pounds. On the thirteenth day it died. Hemorrhage limited to the right lateral ventricle was found. A second case was a twin born at the eighth month. It weighed 4½ pounds. The rectal temperature was about 87° F. It was placed in a couveuse. After the fourth day the temperature ranged between 96° and 98° F. It died on the thirty-seventh day. Hemorrhagic softening of the whole right hemisphere was found.

Rolly[3] reports a case of cerebral palsy **due to syphilis** in twins, reports the clinical history and postmortem findings, and suggests that this etiologic factor may more frequently be in operation than the literature would indicate.

Paralysis after Whooping-cough.—Simonini[4] reports 2 cases of cerebral paralysis occurring in children in the course of whooping-cough. The first child, in the fourth week of pertussis, after a severe bout of coughing became comatose and was found to be suffering from complete hemiplegia. The second child, at the end of the third week, after a severe attack of coughing became convulsed, developed right ptosis, slight paralysis of the right face, and paresis of the left arm. An hour later swallowing was difficult and speech could not be understood. The next night coma, stertor, and Cheyne-Stokes respiration set in, and the child died. At the necropsy punctiform hemorrhages were found in the median and inferior part of the bulb and a fairly large hemorrhage in the anterior median line from the left spinal artery near its origin from the vertebral.

Valentin[5] reports the results of investigation into **paralysis in whooping-cough.** Paralysis in whooping-cough is rare. It affects children especially between the ages of 2 and 6 years. Out of Valentin's 83 collected cases, 62 were of cerebral character. Of these 62 cases, 40 were hemiplegic, 16 were monoplegic, and the rest were paraplegic. The ages varied from 1 to 12 years. A large proportion occurred between the second and sixth year, and it was more frequent in girls than in boys. The rare psychoses attending whooping-cough incidentally noted were delirium with hallucinations, apathy and imbecility, probably of a hemorrhagic and inflammatory nature. Hysteric and choreiform attacks were also noted. The convulsive attacks produced by whooping-cough included cramps and spasm of the glottis, sensory disturbance, embracing blindness, deafness, and anesthesia, and, as extreme rareties, disseminated sclerosis and Friedreich's disease followed whooping-cough. Necropsies showed that the cerebrospinal lesions were small hemorrhages and softenings from the increased blood-pressure and violent congestion. The prognosis was grave as only 40 % recovered from the paralysis.

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[1] Jour. Ment. and Nerv. Dis., Nov., 1902.

[2] Ann. de Gynec. et d'Obstet., Sept., 1901.

[3] Deut. Zeit. f. Nervenheilk., Oct., 1901.

[4] Rif. Med., Nov. 14, 1901.

[5] Paris Theses, 1901.

Thrombosis of the Cavernous Sinus.—E. W. Dwight and H. H. Germain[1] report 4 cases of thrombosis of the cavernous sinus with one cranial operation. In the operated case the patient's condition was decidedly better at the end of the surgical work than at the beginning, the edema and exophthalmos having immediately subsided, and at the end of half an hour the exophthalmos had entirely disappeared, the temperature dropped to 101° F., and the patient was much more quiet. However, he failed, and died 6½ hours after the operation. It has been recognized, say the authors, that the rational treatment consists in opening the cavernous sinus directly, but this has been attempted only once in addition to the case reported, owing to the grave difficulties attending the operation. Hartley demonstrated that it could be done without grave risk provided the patient was in fairly good condition. In the first case the patient lived several months and died as a result of sarcoma. In this case, which was one of forlorn hope, the condition was not made worse by the operation.

NEUROSIS DUE TO INFECTION.

Chorea.—L. Launay[2] in the consideration of the **chorea of pregnant women,** after a description of choreiform jerks, etc., notes the frequency of echolalia and coprolalia in this variety of chorea. He insists that there is a parallel between this disease and hysteria. Usually it is attended by backache, ovarian hyperesthesia, globus, and laryngeal disturbances. He concludes that a considerable number of cases published in medical literature as cases of chorea gravidarum belong to hysteria, that others are maladies des tics, and that others are chronic chorea of the Sydenham variety lighted up or reexcited by pregnancy. Finally, that after excluding all these forms there remain true and incontestable cases of chorea of pregnancy, identical with Sydenham's disease, its peculiar features being due to its occurrence for the first time during pregnancy.

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[1] Boston M. and S. Jour., May 1, 1902.

[2] Thèse de Paris, 1901.

H. N. Moyer[1] contributes an article on the **psychosis of chorea.** He concludes that a well-marked variation of character and mentality may be noted in the majority of cases of chorea, usually for some weeks preceding the movements; that distinct hallucinations are present in a considerable number of cases; that decided mental disturbance in chorea usually comes on after the choreic movements, but may precede them; that the type is usually maniacal, but melancholia or acute delirium may be present; that mental disturbances are commoner in older children and are rarely observed before the twelfth year; that chorea with mental disturbance in later life is almost always associated with organic changes in the central nervous system; that the prognosis is favorable in the mental disturbance of acute chorea of Sydenham, while insanity associated with chorea in middle or advanced life has a decidedly unpromising outlook through association with organic disease of the central nervous system. The author favors the clinical description of Regis, and emphasizes the defects of memory and attention, the mobility of ideas, the lack of consistency in mental recollections, and the mental hebetude; the occurrence of special hallucinations, usually of vision, very rarely of taste or hearing; their occurrence in females, particularly about the age of 14; their appearance in the evening in the drowsy condition between waking and sleeping; their sometimes painful and terrifying nature, causing nightmare and night-terrors; and by their continuance in sleep persistence of the dream state into waking moments.

Henry Thomas[2] gives a short review of the embolic theory of chorea and reports a case in which embolism of the central artery of the retina occurred. The **eye complications** of chorea, the author states, are not numerous. The eyeballs may rarely be the seat of choreic movements, and, as Gowers states, may cause momentary diplopia, which ordinarily is not complained of. The pupils have been described as dilated, as acting sluggishly or not at all to light—a statement which has been confirmed of late. Choreic movements of the iris have also been described, the pupil varying between a pinhead contraction and maximum dilation. Optic neuritis is a very uncommon occurrence, but usually of slight degree, subsiding with the chorea. Schmidt-Rimpler has reported atrophy of the optic nerve, and other cases of this sort are presumed to have followed embolism of the retinal artery or to have been associated with other disease than chorea. Embolism of the central artery is spoken of as extremely rare; the author has been able to collect less than a dozen cases from the literature, to which he adds his own.

P. Guizetti[3] describes a second case of chorea with **septic findings** upon postmortem examination, consisting in a pyemia marked by the presence of Staphylococcus pyogenes aureus. He found a fresh endocarditis, abscess in the spleen, and old ulcers in the colon. From the brain, blood, spleen, and heart numerous colonies of Staphylococcus aureus were obtained. In the brain there was small-celled infiltration with inflammatory foci containing the staphylococcus, and these foci were located deeply in the cortex and the white matter. A number of embolic softenings were also found.

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[1] Jour. Am. Med. Assoc., Nov. 23, 1901.
[2] Johns Hopkins Hosp. Bull., Oct., 1901.
[3] Rivista sperim. di freniatria e medic. leg. delle alien. ment., XXVII, 1901.

M. Reichardt[1] reports 2 cases of **chorea terminating fatally** and examined postmortem, one 4 and the other 20 hours subsequent to death. A very careful examination was made by the most recent methods. A circumscribed inflammation, infiltration of blood, and degeneration of nerve-tissue was demonstrated. The inflammatory process was characteristically perivascular and was found throughout the entire brain-substance. Bacteriologically, the first case was entirely negative; in the second it was doubtful. In the first case the blood from the heart showed Staphylococcus aureus, and in the second case the streptococcus was discovered. The microscopic picture is that of an encephalitis.

Okada[2] records a case of chorea in a girl of 12 with onset 6 weeks after **an acute attack of rheumatism** and 2 weeks after the first signs of cardiac implication. Bronchitis and double pneumonia led to a fatal termination shortly after the commencement of the chorea. Necropsy was made 4 hours after death, which was found to be due to double pneumonia with staphylococcal endocarditis and pericarditis. Thrombi both colorless and red and mixed with hyaline bodies were found in the central nervous system, particularly in the cerebrum. There was a deposit of clotty masses in the adventitia of a medium-sized vein in the white area, and numerous fat-globules in and around the cerebral vessels. A considerable amount of sensory disturbance had been present in the case, associated by the author with the multiple thrombosis. He considers the symptoms of chorea to be due to disturbance in the brain as a result of toxemia, and regards the thrombosis as the extreme expression of these changes. The condition of the nerve-cells is not detailed.

H. Bischoff[3] reports **a senile case,** a patient of 73 years who had suffered from **kidney disease** for several years and presented decided arteriosclerosis, twitching of the head, face, mouth, tongue, the left arm, and of the leg, which during 4 weeks of observation increased and was followed by delirium. During sleep the choreic movements ceased. Finally the patient died. Macroscopically and microscopically nothing positive was found. The author makes a tabular arrangement of cases from the literature, and from it draws the following conclusions: (1) Men and women present about an equal number of cases of senile chorea; (2) rheumatism and heart lesions are rare complications, heart lesions being present in about 12 %; (3) in 60 % the mental conditions are normal; (4) the right and the left side are affected with about equal frequency; (5) 20 % of cases get well, the latest within a year; (6) the anatomic findings do not explain the character and location of the disease.

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[1] Arch. f. klin. Med., 72.

[2] Mitteil. der Med. Facult. der kaiserl. Japan. Univ. zu Tokio, 1902.

[3] Arch. f. klin. Med., 69.

Tetanus.—Coe[1] reports two cases of tetanus **with no assignable cause.** They occurred in hospital practice in New York city, both in his wards. The first operation was the removal of a double salpingitis and cystic ovaries; no complications. Stiffness of the neck was felt on the twenty-first day and death occurred on the thirty-sixth day. The second patient had hysterectomy and did well until the ninth day, when stiffness of the jaw set in, and on the tenth day she could not swallow. Fifty cubic centimeters of antitoxic serum was injected into the veins, and a second injection was made the following day. Death occurred on the eleventh day. Both patients were hysteric, so the diagnosis was obscure.

B. Moellers[2] claims that the use of **antitoxin injection** later than 6 hours after the beginning of symptoms is practically useless, and even then must be employed in extraordinarily large quantities to effect any advantage. He believes that the most important application of antitoxin rests in its use at once as a prophylactic in wounds soiled in such a way as to make it possible for tetanus to develop. In his cases, which were severe ones, large quantities of antitoxin were injected early, yet the cases resulted in death, without any benefit attributable to the treatment.

Tetany.—Solovieff[3] reports 2 adults suffering from tetany who showed violent **rhythmic contractions of the diaphragm,** perceptible both by inspection and palpation and synchronous with the heart-beat. These contractions were attended with a slight whistling sound due to the entrance of air into the left lung, which was sharply inflated at each contraction of the diaphragm. These spasmodic contractions were verified by the radiograph and produced, according to the author, by irritation of the phrenic nerve through the movements of the heart. They ceased during free and deep inspiration. The author proposes the term the **phenomenon of the phrenics.**

Bassal[4] has collected certain facts showing that an **infant at the breast may suffer from tetany when the mother or the nurse is the subject of exophthalmic goiter.** The condition apparently is due to poisonous materials formed in the system of the nurse and so modifying the milk. In ordinary goiter mother's milk does not seem to have any ill effects. Thus in certain sections where goiter is frequent tetany is exceptional, but it is thought that, attention having been directed to the matter, a more common association of tetany with goiter in the mother may be observed.

TROPHONEUROSES.

Graves's Disease.—J. A. Booth,[5] considering the **radical cure** of exophthalmic goiter, concludes: (1) Cases of Graves's disease may be completely cured both by thyroidectomy and bilateral section of the sympathetic. (2) In view of the fact that some cases are cured by internal medication, there must be a certain proportion in which the affection does not induce structural changes in any organ. (3) No theory can be

[1] Am. Jour. Obstet., Sept., 1901.

[2] Deut. med. Woch., Nov. 21, 1901.

[3] Rousski Vratch, 1902.

[4] Thèse de Toulouse, 1901.

[5] Jour. Nerv. and Ment. Dis., Sept., 1902.

regarded as adequate without taking into consideration the function of the thyroid gland. (4) Three factors must be considered in the production of the disease: (*a*) The central nervous system; (*b*) the connecting fibers, sympathetic and vagus; (*c*) the thyroid gland. (5) A lesion of one of these parts may produce a specific alteration in the others, the consequences of which, together with the exciting cause, may give rise to the symptoms of Graves's disease.

Varcot and Roy[1] report a case of exophthalmic goiter in a boy of 4½ years, with the characteristic triad of symptoms.

Schultes[2] reports a case of Graves's disease **treated with the serum of dogs** from which the thyroids had been previously excised, following the plan of Ballet and Enriquez. The patient, a female 49 years old, had suffered from symptoms of exophthalmic goiter for 4 years. Palpitation and nervous symptoms had greatly increased in severity for 3 months, and for 6 weeks she had mental disturbance, reaching a climax on the day before admission to the hospital, when she had delusions and hallucinations and took very little nourishment. The exophthalmos was marked, the thyroid much enlarged, cardiac dulness increased to the left, pulse 140, muscular weakness pronounced. At first a half gram of the serum of sheep from which the thyroid had been removed was given by the mouth 3 times a day; later the preparation prepared by Merck under the name of antithyroidin, and the dose was daily increased until she took 4 grams 3 times a day. No other treatment was employed. After 15 days of the treatment she had become mentally clear and was quiet. After 24 days she was free of symptoms of psychic disturbance, pulse 100, and a week later the circumference of the neck was reduced a half inch and the pulse had fallen to 88. The goiter was generally softer, a few hard nodules remaining. She was able to sew, which had formerly been impossible on account of the tremor. The thirty-ninth day of treatment was marked by some palpitation and increase in the size of the goiter, but this was temporary. She was discharged after forty-nine days as well, and Schultes ascribes the short duration of the psychoses to the treatment.

S. W. Thompson[3] records an interesting case of Graves's disease **attended by Raynaud's symptom-complex** which reached a degree resulting in local gangrene and death, causing exfoliation of several fingers and the palmar surface of the hand. The discomfort and pain in the hand grew so intense that the patient became addicted to morphin, which he took to the amount of 24 grains daily by the hypodermic method. On this ground, also, to relieve the pain, the arm was amputated at the lower third of the humerus. In spite of this the patient was extremely melancholy, and threatened suicide, and on the day after the operation refused food or drink, became delirious, grew weaker, and died several days later.

C. L. Dana[4] reports **an acute case with early fatal termination.**—The patient was an unmarried female of 23. Nothing notable in the family history. The disease had apparently been present about a month before her admission to the hospital. Seventeen days subsequently she passed into a stuporous condition and it was found she could not swallow. Then speech was abolished; there was some paresis of the right side of the body and ptosis of the left lid, leading to the belief that there was a lesion of the pons near the left crus. Postmortem examination showed no gross cause of death. The viscera were normal. The thyroid was enlarged with some hyperplasia. The thymus gland was also enlarged. The membranes at the base of the brain were congested; there was some excess of fluid, but no evidence of inflammation. Upon carefully examining the base of the brain an area of softening was found involving the left crus and part of the pons adjacent. This softening showed no hemorrhage. In a second case, of a chronic variety, changes in the nuclei of the bulbar nerves were made out. Sections of the medulla at the level of the tenth, twelfth, and ninth nuclei showed very interesting changes. Cell-bodies were pigmented even to the point of destruction of the cell; vacuolation was frequently observed. [The cases are important as showing bulbar lesions both in the acute and chronic variety of Graves's disease.]

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[1] Bul. de la Soc. de Pediatrie de Paris, June, 1901.

[2] Münch. med. Woch., May 20, 1902.

[3] Med. Rec., Oct. 11, 1902.

[4] N. Y. Med. Jour., June 14, 1902.

Abadie and Collon[1] report favorable results in the treatment of Graves's disease by the **injection of a mixture of iodin and ether** into the large gland, a plan first recommended by Pitres in 1899. It is stated that of 12 patients, 6 were cured and the rest improved. Twenty-four cases have been treated by the authors. To 20 grams of ether 4 grams of iodoform is added, and of the solution 1 cc. is injected per dose into the largest part of the affected thyroid gland. A notable diminution of the thyroid occurs at the time of injection. There is usually a sense of swelling of the neck and slight choking; pinching and darting pains are sometimes felt in the neck, jaw, face, and ear, or there may be a slight cough. Inflammation does not follow the injection, and only once was there any untoward symptoms—namely, fanting. When partial improvement occurred, injections were given at intervals of a few days until improvement was complete. Improvement consisted in the disappearance of headache, agitation, irritability, amenorrhea, polyuria, neuralgic pains, then the rapid heart subsided, attacks of angina, tremor, and the exophthalmos finally disappeared, appetite improved, and the strength returned. The general results of treatment were that 12 cases were cured, 9 showed notable and permanent improvement, and 3 temporary improvement. The injections were always made under strict antiseptic conditions.

Tedeschi,[2] experimenting on dogs, finds that if the **restiform bodies** are injured just behind the acoustic tubercle most of the symptoms of exophthalmic goiter appear—namely, exophthalmos, enlargement of the palpebral opening, tachycardia, generalized tremor, sometimes polyuria, glycosuria, and salivation.

The Thymus Gland.—Stokes, Ruhrah, and Rohrer[3] discuss the relation of the thymus gland to marasmus, and conclude: (1) Atrophy of the thymus gland is always found in cases of infantile atrophy; (2) the condition of the thymus gland is an index of the general nutrition in infants; (3) the state of nutrition of infants may be estimated by a microscopic examination of the thymus at autopsy.

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[1] Revue Neurol., Jan. 30, 1902.

[2] Gaz. degli Osped., April, 1902.

[3] Am. Jour. Med. Sci., Nov., 1902.

Raynaud's Disease.—H. Cushing[1] reports the use of the **tourniquet in the treatment** of vasomotor spasm of Raynaud's disease, and calls attention to the familiar observation that after the use of Esmarch's bandage an exsanguinated limb rapidly fills with blood. This observation was applied to the treatment of the blanching in the extremities in Raynaud's disease. He found the treatment simple of application and presenting considerable success in the instance in which he had the opportunity of trying it. If employed early enough or repeated with sufficient frequency, his case suggests that the underlying cause of the spasm either subsides, or, what is more probable, the local vascular condition becomes so altered by the periods of active circulation that peripheral stimuli no longer provoke the constrictor responses resulting in local asphyxiation. The treatment would be considered palliative, and of course one of decided limitations.

G. S. C. Badger[2] reports **a pronounced case** of this disorder in which a woman of 45 had suffered mutilation of fingers and toes.

Emil King[3] reports an interesting case of Raynaud's disease presenting **difficulty in its early diagnosis.** The patient experienced much more pain than is commonly presented, and this, with the physical appearance, strongly suggested erythromelalgia. Lung symptoms and sudden vertigo apparently were due to vasomotor irregularity within the brain and lung. The author insists that cases such as this assist in proving that the disease is by no means confined to the extremities.

Angioneurotic Edema.—T. F. Raven[4] reports a case of nervous edema of the tongue in a woman 86 years of age otherwise in good health. The swelling was confined to the right half of the tongue. Later she suffered another similar but more extensive attack.

T. W. Griffith[5] records **a family form**—a young woman of 16 who ever since infancy had been subject to localized swellings on various parts of the body. Shortly after birth one arm swelled to such an extent that the clothing had to be cut to release it. She often noticed a small swelling like a marble which would become diffuse so that the skin on the back of the hand would swell enormously and with great rapidity. The usual locations were the hands, feet, shoulders, chest, and genitals. Sometimes there was slight redness and tingling; occasionally, when the vulva was affected, urination was painful. Her father from earliest infancy had presented swellings of the same kind. On 3 occasions they affected his throat, and the third attack caused his death at the age of 29. The daughter died from a similar involvement of the larynx, dying like her father in convulsions from suffocation. Both deaths were witnessed by the mother, who said that they were identical. Postmortem examination in the girl's case revealed a very edematous mucous membrane in the larynx entirely occluding it. The edema affected not only the mucous membrane, but the deeper connective tissue, and even the substance of the muscles.

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[1] Jour. Nerv. and Ment. Dis., Nov., 1902.

[2] Boston M. and S. Jour., Jan. 30, 1902.

[3] Amer. Med., May 17, 1902.

[4] Brit. Med. Jour., Dec. 21, 1901.

[5] Brit. Med. Jour., June 14, 1902.

Akromegaly.—S Kuh[1] claims that inasmuch as akromegaly is always associated with disturbance of the pituitary body, natural treatment might consist in feeding these patients with this element. He has tried this in 3 cases. The first derived little if any benefit, while the other two cases, after 1½ years and 1 year respectively, seem decidedly improved; headache, dizziness, general weakness, sweating, and vomiting disappeared The trophic changes in the nails of the second patient became much better, and the beneficial changes he attributes to the use of the pituitary gland. [It may be said that these results do not correspond with those obtained by other observers. They are suggestive, however, and this line of treatment should not be omitted in the care of those cases which are not amenable to any other line of treatment. The exact relation of the pituitary body to the disease is perhaps still open to discussion. We know a case in which akromegaly was not accompanied by disease of the pituitary body, although in this particular case the conformation of the patient might perhaps call the diagnosis into question.]

Israel[2] reports a case of akromegaly under observation for 3 years by Gerhardt. The patient died from gradually increasing mental and bodily feebleness. Section discovered the **hypophysis to be no larger than normal** and showed neither macroscopic nor microscopic changes. The enlargement of the skeleton was remarkable. There were numerous supracartilaginous exostoses, and periosteal and parosteal hyperostoses. The soft parts did not show the changes commonly described in akromegaly. The thyroid gland was small and there were no remains of the thymus. [This case is of great importance, as many claim that pituitary tumor is an essential finding in akromegaly.]

The Pituitary Gland.—A. Caselli, in an anatomic and experimental study, published in Italy in 1900, contends that the posterior lobe contains no nervous elements, or at least only elementary ones. That the complete abolition of the hypophysis in dogs and cats causes at first slowing of the respiration and acceleration of the pulse, then mental depression and disturbance of movement, such as arching of the back and spastic gait, without tonic or clonic contractions of the limbs; afterward progressive cachexia sets in, and the animal dies comatose. The cachexia, according to the author, is due to intoxication and the mental disturbance to alteration in the cerebral tissues brought about by the intoxication, and the motor disturbance depends upon similar lesions in the spinal cord. The extirpation of the hypophysis gives rise to diabetes through injury to the part of the brain near it. Removal of the hypophysis modifies the course of the tetany of parathyroidectomy and causes the motor disturbance to be replaced by paralysis, soon followed by coma and death. In dogs deprived of their thyroid gland extirpation of the hypophysis increases the cachexia without altering its character. The hypophysis appears to be analogous to the thyroid but unable to fulfil its functions. Structural alterations of the hypophysis increasing its size give rise to disturbances dependent upon injury to the optic nerves through raising of the intracranial pressure. Morphologic alterations consist partially in hypertrophy, giving rise to akromegaly. He also finds that pituitary extract is applicable as a therapeutic agent in cases of mental disease in which there is depression. [In the experiments upon which these findings were based care was exercised not to confound the symptoms of removal of the hypophysis with disturbance of the meninges in the brain, but it is impossible to feel sure that part of the results of the operation are not to be attributed to local injury.]

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[1] Jour. Am. Med. Assoc., Feb. 1, 1902.

[2] Virchow's Archiv, vol. CLXIV, 344.

Adiposis Dolorosa.—M. H. Bochroch[1] reports an additional case of adiposis dolorosa, which of late is coming to be designated as Dercum's disease. In this case are presented the 4 symptoms insisted upon by Vitaut—fatty enlargement, tenderness, neurasthenia, and mental symptoms, here assuming the form of impairment of memory and mental hebetude. The pains in this case were both spontaneous and induced, and were more or less constant. The asthenia was decided and the mental symptoms are also typical, although not pronounced. The patient was a woman 56 years of age. The family history presented nothing particularly notable. At the age of 19 she weighed less than 100 pounds. She had 8 children, 5 of whom are living, 1 being feeble-minded. One died in infancy of cholera infantum, another of marasmus, another of diphtheria. Shortly after marriage she noticed an accumulation of fat over the hips and upper part of the arms and legs. She passed the menopause at the age of 50. For the past 7 or 8 years she had observed ready bruising and intense pain caused by the slightest blow. For 2 years she had suffered from sharp shooting pains in the arms and hands and was treated for neuritis. At the date of the report, in 1902, she weighed 229 pounds. She had marked traces of arteriosclerosis with numerous fatty deposits, one under the chin being the size of an egg. The proximal portions of the limbs and trunk are particularly affected, folds of the fat from the abdominal and loin region lap over the crest of the ilium. [Unfortunately nothing is stated as to the woman's habits, the possibility of alcohol or the suspicion of venereal poisoning as to the etiologic medium in the case.]

Dercum and McCarthy[2] report further upon a case of adiposis dolorosa previously published and give the **autopsical findings.** The patient showed the characteristic contours of the condition. A résumé of the pathologic findings is as follows: Adenocarcinoma of the pituitary body; anomalous arrangement of the cerebral convolutions; excessive fissuration and confluence of fissures; fibroid hemolymphoid nodules in the subcutaneous fat, in a state of acute congestion; interstitial neuritis of the nerve-filaments of the superficial fat; telangiectatic angioma, with slight interstitial hyperplasia of the spleen; defective development of the testicles; acute parenchymatous nephritis; cutaneous erysipelas. Just what may be the rôle of the thyroid gland in this disease is problematic. The cases reported by Dercum and Burr would lend color to the assumption that the changes in the thyroid gland, being atrophic in character, are related to the excessive production of fat. In the present case, however, changes in the thyroid gland, though present, were not pronounced. The changes in the pituitary body, on the other hand, were very marked; and bearing in mind the interrelation which exists between the thyroid gland and the pituitary body, the changes in the latter are, to say the least, very significant. The pituitary body is thus brought into relation, though perhaps indirectly, with a fat-producing or fat-destroying function, a relation which up to this time has not been considered. The fact that the thyroid gland has been found diseased in every one of the 5 cases coming to autopsy is significant. Thus far 28 cases of adiposis dolorosa have been reported. Both the clinical findings and the results of the autopsies establish the fact that adiposis dolorosa is a well-defined clinical entity. The remarkable character of the post-mortem findings should stimulate pathologic studies in simple adiposis.

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[1] Am. Jour. Med. Sci., Oct., 1902.

[2] Am. Jour. Med. Sci., Dec., 1902.

J. B. Roberts[1] reports a case of this somewhat rare disorder occurring in a French woman 60 years of age, who presented a **peculiar menstrual history.** She weighed 97 pounds when she was married at the age of 20. Her husband, an army officer, was called away 6 weeks after marriage and subsequently killed; she never saw him after he left her. Menstruation, which was taking place at the time she heard of his injury, stopped at once as the result of the shock. Twelve years later she again menstruated once. In 1893, after a fracture of the fibula of the left leg, the lower extremities, which had formerly been of normal size, increased, especially the one that was injured. Legs, thighs, and the region about the anus became extraordinarily enlarged; the buttocks were enormous and contained lumps and masses of fat, and all of the enlarged parts were very painful to manipulation. Although the patient was generally fat, the portions mentioned were extraordinarily involved. The case is interesting because of the menstrual history. Most of these cases have shown some disturbance of menstruation or of thyroid activity or both.

F. X. Dercum[2] reports **2 additional cases** of adiposis dolorosa, and quotes Vitaut as dividing the condition into the nodular, the localized-diffused, and the generalized forms. Both of the cases reported pertain to the second group, both showing a preponderance of deposits in various localities, in one upon the trunk, in the other upon the thighs. Both patients showed the characteristic symptoms of the condition—fatty deposits and pain and tenderness. Crises of pain, sometimes noted, were absent. Vitaut calls fatty deposit, pain, asthenia, and mental phenomena the 4 cardinal symptoms—a statement with which the author is disposed to agree.

Hyperostosis Cranii and Osteitis Deformans.—Morton Prince[3] reports upon several cases of so-called hyperostosis cranii and believes that there is no sure ground for differentiating between hyperostosis cranii and osteitis deformans; that they are both trophic disorders, but that they are very probably allied disorders or only different manifestations of the same disease; that the osteoarthritic changes commonly found in osteitis deformans are not complications; finally, that the autopsies thus far made do not exclude the nervous system as the seat of the trophic derangement; that the changes found in the spinal cord and peripheral nerves suggest a neuropathic origin similar to that of the myopathies.

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[1] N. Y. Med. Jour., April 26, 1902.

[2] Phila. Med. Jour., Mar. 1, 1902.

[3] Am. Jour. Med. Sci., Nov., 1902.

Trophoedema.—Under this title Henry Meige[1] gives a general description of conditions marked by localized permanent edemas, such as those described in the YEAR-BOOK for 1901 under the title "Symmetrical Trophoedema." He finds the condition of chronic trophoedema one characterized by a white, hard, painless swelling, occupying one or numerous segments of one or both lower extremities and lasting throughout life without any material disadvantage to the general health. Sometimes it is encountered singly, but more frequently is hereditary and of family distribution, and may also be congenital. In other instances, however, it may be confined to the upper extremities or limited to one-half of the body and face, as reported by Rapine in the same journal. Mabille also illustrates an instance in which the edema is confined to the lower extremities. A. Thomas would bring congenital hemihypertrophy of the body and extremities into the same list, and reports an interesting case, with illustrations, also in the same publication.

MOTOR NEUROSES.

Paralysis Agitans.—Walbaum[2] relates a case which exhibited **macroscopic changes** in the brain in the form of calcified vessels in the gray matter of the central ganglia while the rest of the cerebral vessels were unaffected. Microscopically he found changes in the nervous elements and glia like those described by various authors and of late credited tc old age, but he is not inclined to agree that they can be looked upon as without distinct relationship to paralysis agitans. He thinks his case gives further proof that paralysis agitans is not due to lesions of the cord, but belongs rather to the class of functional motor neuroses.

Myotonia.—G. Rossolimo[3] illustrates and gives a detailed description of a case of acquired myotonia presenting the **contours of progressive muscular atrophy.** A man of middle age who for 3 years had presented well-marked symptoms of myotonia in the muscles of his face, neck, and trunk and four extremities, most pronounced in the arms. All the muscles affected showed the myatonic reaction and other physical conditions associated with the disorder. Later atrophy was added to the condition, of a progressive musculospinal type, producing the myopathic face and the involvement of the distal portions of the 4 extremities. Similar cases have been reported by Jolly, Pelizaeus, Kornhold, Schoenborn, Sirol, and Hoffman. The muscles affected by atrophy showed, in addition to the myotonic response, quantitative enfeeblement for all forms of current, and, in addition, in some muscles there was a qualitative reaction of degeneration, and all the tendon reflexes were diminished.

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[1] Nouv. Icon. de la Salpêt., Nov., 1901.

[2] Virchow's Archiv, Bd. CLXV.

[3] Nouvelle Icon. de la Salpêt., April, 1902.

Paramyoclonus Multiplex.—F. W. Langdon[1] reports another case of this rare disease, and adds unpublished notes of the further progress of a former case reported by him. The new patient was a girl of 13 complaining of muscular spasms occurring in paroxysms, which had developed 6 days previous to her admission to the Presbyterian Hospital in Cincinnati, preceded by a pain in the back and following a fright. The family history was practically negative. The patient was strong and healthy at birth, was said to have had spinal meningitis at 6 months of age, measles at 4 years, and peritonitis at 10. At 11 had an illness which was diagnosed as St. Vitus's dance, which seemed to resemble the present attack. The patient was undeveloped physically, with defective teeth, suggesting the Hutchinson variety but not typical. The pulse was normal. Menstruation had not appeared. The mental state indicated extreme fatigue. Complains of no pain or discomfort, but says "don't hurt me" when gently moved about. General hyperesthesia to ordinary touch not confined to localities or zones. The muscles of the trunk and those reaching to the extremities are the seat of frequently repeated violent clonic contractions resembling those produced by the closure of a galvanic circuit. They were bilateral and synchronous on the two sides usually, though commonly more vigorous on the left side as far as the limbs were concerned. Transient opisthotonus was produced by the spasms, and several times these were sufficiently violent to throw her from the bed. The spasms are practically continuous during the waking hours and rendered more violent and frequent by manipulations. Under hyoscin hydrobromate and similar sedatives the spasms were gradually controlled and the intervals between spasms were increased until finally none occurred in 48 hours. This is a preliminary report only.

PSYCHONEUROSES.

Epilepsy.—W. P. Spratling[2] reports upon the **causes and manner of death** in epilepsy and emphasizes the contention that epilepsy should not be regarded as a relatively harmless disease as far as life is concerned, for, as a matter of fact, any epileptic may die suddenly at any time. The manner and cause of death in epilepsy he describes as follows: (1) It may occur suddenly as the result of a single seizure; (2) it may occur from a series of seizures ending in status epilepticus; (3) it may result from an accident suffered because of a fit; (4) it may result from all other causes that seemingly produce it, with a seeming preponderance in favor of diseases of the heart and lungs. He notes that in a total of 95 deaths which occurred at the colony for epileptics in New York 23 % were due to status epilepticus. About one-half as many died from accidents during the fit, these taking place generally in bed. He cites the statistics of the Ohio Hospital for Epileptics, which showed that out of 190 deaths 42 were due to diseases of the lungs either in the form of tuberculosis or pneumonia; 22 % of the entire number. He calls attention to the very important need of the supervision of epileptics during their sleep, as they are frequently disposed at the end of the convulsion to roll over upon their faces and smother in a soft bed.

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[1] Jour. Nerv. and Ment. Dis., Sept., 1902.

[2] Med. News, June 28, 1902.

W. P. Spratling[1] states that in the United States 1 in every 500 suffers from epilepsy. In continental Europe the proportion is thought to be somewhat less. He found in 1000 observations that **a rise of temperature** follows 55 % of all attacks, the temperature going highest after the severest seizures in which muscular contractions are pronounced, but also sometimes reaching 102 or over in petit mal and psychic attacks. The temperature, however, is not augmented after every attack. In regard to treatment, the author contends that the epileptic and the disease should be considered as a unit and treated as such. The treatment should be varied to meet the requirements, divided into surgical, medical, dietetic, and moral.

G. M. Gould[2] reports 6 cases of epilepsy **associated with ametropic eye-strain.** The cases are reported with such brevity that they are of comparatively little value, although it appears that most of them were carefully observed, and the history has extended over a sufficient length of time to indicate not only that the eye-strain was an essential feature, but that its correction led to a practical cure. The author takes a very proper position in reference to the relation of eye-strain and epilepsy, and indicates that in exceptional cases the ocular condition may be the pernicious factor which maintains the epileptic manifestations. The obligation is imperative that in every case of epilepsy the condition of the eyes should be carefully considered.

A. Ferenczi[3] examined 33 cases of epilepsy in reference to the **knee-jerks.** In 22 he found the reflex increased during the attacks, and 6 times it was normal in the postepileptic period. Only 6 times did he find the reflex lessened or absent. The sign was sought in 158 attacks and its condition was entirely relative to the general muscular tone.

Schaefer[4] gives his experience with the **feeding of epileptics** after the manner of Toulouse and Richet, consisting principally in the withdrawal of salt. He records several cases in which decided benefit seems to have followed the régime, the attacks occurred at longer intervals, were less severe, and the mental state, which had been seriously disturbed, improved.

G. Garbini[5] reports that **Richet's method** was tried on 14 adult epileptics presenting severe symptoms and an unvaried character of the disorder. For a considerable period they had received 8 grams of bromid daily, without apparent benefit. The bromid was discontinued for a month; then for a month, without change of diet, 3 and after a week 4 grams of bromid were employed daily. In the third and fourth months the diet was deprived of salt the first week of this régime, in the second, third, and fourth week one gram of bromid was used daily, and the last

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[1] Jour. Am. Med. Assoc., May 3, 1902.

[2] Amer. Med., July 5, 1902.

[3] Orvosi Hetilap, 1901, No. 33.

[4] Neurolog. Centralbl., Jan., 1902.

[5] Rivista mensile di neuropatologia e psichiatria, 1901, No. 8.

4 weeks no bromid was given. The results were as follows: with a diet poor in salt and one gram of bromid the number of attacks fell 73 %. The same diet without bromid secured a reduction of 65 %. The duration of the attacks, as a rule, was reduced two-thirds in point of time. The general condition of the patients was improved.

The question of **diet in epilepsy** is afforded considerable light by H. Schloss.[1] His experiments were made upon 16 patients, divided into 4 groups. The first 2 groups were given an exclusively meat diet for 6 weeks and an exclusive milk and vegetable diet for another 6 weeks, while groups 3 and 4 received milk during the first period and meat later. He found that neither diet exercised in the respective groups any appreciable effect upon the number of attacks, the general nutrition of the patients also remaining unaltered. He then chose a diet very poor in common salt, and found that he could diminish the intake of salt to 2.1 grams, the caloric value of the food being kept up to 2387. At the same time, the patients received 4 grams of mixed bromids. Four out of 5 patients on this regimen lost weight, and the diet was increased to 3243 calories. All 5 patients still lost in weight. The diet was then increased to 3760 calories. The patients still lost in weight, becoming weak and pale. At the same time, the number of attacks diminished, in 2 ceasing altogether, while in 3 they became of rare occurrence. Psychic disturbances which had previously occasionally appeared were unaffected. He next investigated the effect of fat and acid foods, without being able to detect any influence on the number of attacks. In the consideration of the use of alcoholics in a series of cases he allowed the patient 0.5 liter of beer every evening, but no effect seemed to be induced by this. He then allowed 0.5 liter at dinner as well as supper, without any noticeable effect upon the attacks.

Hebold and Bratz[2] have carried out a number of valuable experiments **relative to autointoxication in epilepsy.** They injected the urine and blood of epileptics into dogs and white mice, and conclude that these experiments fail to show any connection between the toxicity of the juices of the body and the occurrence of epilepsy.

M. C. Thrush[3] reports upon the special value of **solanum carolinense** in the treatment of epilepsy. This was used in a number of cases observed in the Philadelphia Hospital with, for the author, encouraging results. The fluid extract of fruit or root as prepared by the author was used, and also found to be useful in hysteroepilepsy. Usually it acted better than the bromids. The preparation to be used is the freshly prepared fluid extract given in ascending doses from 1 fluid dram. No toxic symptoms followed its administration and the mental faculties are not impaired by its use.

W. H. Thompson[4] contributes a very interesting article upon the **pathology and treatment** of epilepsy, without adding anything particularly new. His masterly grasp of the situation would invite perusal of the entire contribution. Medicinally, the bromids of course occupy first place, but the great factor in the treatment of epilepsy is prophylaxis. To the mere routine administration of drugs and the undue reliance upon them is due the chief want of success. In this connection the author directs attention to the toxic and toxemic states which, common to all individuals, are especially likely to develop in epileptics. The author concludes that it is the gastrointestinal tract which is the commonest source of such autointoxications, and these cases show the common factor of accumulated toxic influences followed by explosive manifestations, just as is seen in uremia, gout, rheumatism, migraine, etc. The condition of the intestinal tract, therefore, deserves first notice, but a close second is furnished by the functions of the kidneys. Of the first importance is outdoor life and abundant exercise or work. After an experience of 30 years this sanguine author states that the prognosis for cure in acute cases is quite 70 %, and the chief reason why these figures are not ordinarily reached is the difficulty in getting the patient to persevere in the whole details of the essential treatment.

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[1] Wien. klin. Woch., No. 46.

[2] Deut. med. Woch., No. 36, 1901.

[3] Phila. Med. Jour., May 3, 1902

[4] N. Y. Med. Jour., Nov. 15, 1902.

Rushton Parker[1] reports a case of **traumatic epilepsy** in which there was adhesion between the scalp and the brain, cured by opening the old wound and inserting a plate of gold to prevent further adhesions, and closing the wound.

Myoclonus Epilepsy.—L. P. Clark and T. P. Prout[2] contribute an interesting and valuable article on the subject of **myoclonus epilepsy**, or the **association disease.** They believe that while myoclonus is a rare affection, the association disease is still more rare, only 57 cases so far having appeared in the literature, including 4 reported in this article. The **predisposing causes** of the association disease are family degeneracy, which equals that of essential epilepsy, and the association disease also often appears in family groups. Physical degeneracy or mental stigmas of degeneracy are almost invariably present, and the disease appears to be directly transmissible. The immediate excitant of the disease is usually trivial, and therefore probably of no significance. Men are more frequently affected, about 5 to 3, as far as reported cases indicate. All cases have developed in early life—that is, under the age of 20—except one. In three-fourths it appeared between the ages of 9 and 15 years, closely resembling epilepsy in this respect. The symptoms of the association disease are usually those of epilepsy first; the convulsive attacks appearing in one-half the cases antedated the myoclonus from a few weeks to several years. In but one-third of the cases did myoclonus appear first, and in the remaining cases both diseases appeared at once. Sometimes the epileptic paroxysms are extremely rare, as in one case in which only two attacks of grand mal appeared during the life of the patient. The seizures are never preceded by sensory aura, and in fact no aura at all, but premonitory increase in myoclonic contractions are usually in evidence, and are followed at their climax by epileptic seizures, although these may not take place, the entire attack being aborted. The tonic stage of the fit is sometimes much curtailed; tongue-biting and postparoxysmal coma are also absent. The muscles mostly involved

[1] Brit. Med. Jour., May 24, 1902. [2] Am. Jour. of Insanity, Oct., 1902.

in the epileptic seizure are those engaged in the myoclonus and no periodicity marks the occurrence of seizures, and no myoclonus epileptic is reported as dying of status epilepticus, although a sort of status myoclonus frequently hastens death and may actually produce it. The mental automatism associated with epilepsy is of frequent occurrence in the association disease. Melancholic mania is also frequently encountered. These patients have long series of bad days. The mental stigmas of epilepsy are usually present. Myoclonus of the association disease is somewhat atypical compared with the essential myoclonias. The contractions are usually lightning-like, but may have a fibrillary character, involving certain parts of muscles only—a condition described as "live flesh"—and such manifestations are likely to develop into typical myoclonic contractions, though they may remain fibrillary through the life of the patient. A single general tonic contraction may rarely constitute the entire clinical picture. Myoclonus contractions end imperceptibly in the tonic stage of the epileptic paroxysm. The contractions are often strong and affect large masses of muscles, rendering locomotion difficult. The trunk is frequently affected, causing the body to jerk backward and forward and laterally. The face and distal portion of the extremities are frequently involved. Generally the myoclonus symmetric, but both sides are not always synchronously involved. Commonly it begins in the upper extremities, and in a few days or weeks involves the lower extremities, the body, neck, and face in the order given, the muscles about the eyes and mouth being the last affected. The tongue and diaphragm frequently suffer, in severe cases producing grunts and barks, a sort of laryngeal tic. As the malady develops the myoclonus becomes more and more persistent during the waking state, having a tendency to develop the myoclonic status, which may terminate in death. As a general rule, superficial and deep reflexes are increased, and also, as a rule, there is lack of physical and mental development. Rachitis, lack of muscular and bone development, poor nutrition, anemic and gastro-intestinal symptoms, and albuminuria are frequently encountered. Progressive dementia is an added feature in some cases. The **prognosis** as to recovery is extremely poor. Those who have reached advanced years present extreme senility and progressive dementia. Individuals thus afflicted with the association disease are commonly sterile or children born of them die of intercurrent affections, such as eclampsia, tuberculosis, and meningitis. The **diagnosis** in a typical case is easy. There are family types and sporadic cases, the majority being of the latter variety. The condition may be acute and severe or mild and chronic. The differentiation is from hysteroepilepsy, essential epilepsy, post-hemiplegic chorea associated with epilepsy, choreic epilepsy, and general paresis. The **treatment** can be only palliative, but, as a general rule, long remissions both in the myoclonus and the epileptic course may be obtained by the use of bromids. Surgical intervention is contraindicated. The general treatment is the same as that of epilepsy. As to the pathology, the location of the disease has generally been considered as cortical. This contention is confirmed by the authors. There have been 7 autopsies, of which 2 were made by Clark and Prout. The literature gives no report upon the microscopic appearance of the cortex, but in one case carefully studied by Clark and Prout lesions which they considered characteristic of epilepsy in the so-called sensory cells of the cortex were found; that is, they recognized the same changes which they commonly find in the second cortical layer of cells in epilepsy plus similar changes in the large pyramidal cells of the third layer. They infer that the excitant is probably a general toxic agent acting in a uniform manner upon these cells, inducing the changes in question, but coupled with an inherent organic anomoly, making these cells primarily vulnerable. Case-histories of 4 patients are detailed at length and the autopsical findings in 2 cases.

Hysteria.—W. G. Spiller[1] attaches importance to **the lacrimal reflex** in the diagnosis between organic and hysteric anesthesia of the face. He quotes Giles de la Tourette as saying: "Each time we detected anesthesia of the ocular mucous membrane by direct contact with a piece of paper, for example, the tears were immediately secreted, and in the same abundance as with the other eye from the same excitation." The author has examined a case in which the sensibility had been destroyed by operation upon the right gasserian ganglion. He found that irritation of the nostril was the most satisfactory way of testing the lacrimal reflex. By irritating the nostril on the side of the anesthetic area with a probe no increase in the secretion of tears could be seen on either side; but if the opposite eye was irritated, the left eye lacrimated freely without increase of lacrimation on the affected side. Small pieces of paper placed between the lid and eyeball of the affected side caused very little increase of secretion in either eye, but when placed upon the sound side the lacrimal secretion was increased on that side considerably, but very little, if at all, on the affected side. In another, similar case, in which the gasserian ganglion had been removed, there was also a distinct difference in the amount of secretion on the two sides. The author therefore infers that when one side of the face is anesthetic the retention of the lacrimal reflex would suggest a hysteric condition.

The **ear symptoms** in hysteria are very fully set forth in a monograph by Chavanne.[2] He concludes that the hysteric ear manifests itself in two ways: first, as an epiphenomenon; second, as a monosymptomatic hysteric state. The first being frequent and the second rare. In every instance before asserting the hysteric nature of the symptoms it is essential to determine the nonexistence of any lesion capable of explaining the phenomenon. The hysteric otitic syndrome he would generalize as follows: (1) Sensibility of the ear and of the cartilaginous portion of the auditory canal follows conditions of sensibility on the same side of the face; (2) sensibility of the bony portion of the canal and the drumhead has an independent relation; (3) absolute anesthesia of the drumhead is exceptional, and the same is true of the tympanum and the eustachian tube; (4) different portions of the ear may present hyperesthetic features and furnish hysterogenetic zones, though these are rare; (5)

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[1] Phila. Med. Jour., May 17, 1902.

[2] Oreille et Hysterie, Paris, 1901.

acoustic sensibility does not constantly correspond to the cutaneous sensibility; (6) the diminution of bone conduction is constantly greater than that for air conduction; (7) Rinne's test is always positive.

Mental Symptoms of Neurasthenia.—W. K. Walker[1] emphasizes the following points: Every case of neurasthenia presents mental symptoms in some form. The difference between neurasthenia and certain forms of mental disease is largely one of degree; in fundamental nature they are the same, *i. e.*, a pathologic lowering of the psychic functions—having the same etiology and, so far as we have yet been able to ascertain, the same underlying pathology of nutritive change in nerve-cells. Viewed in this light, the mental symptoms assume greater significance, in many cases leading us to anticipate a greater degree of defectiveness to follow, depending upon duration and virulence of exciting causes, the peculiar nature of causal poisons and vulnerability of individual tissue; and when they are encountered in one of previous known mental weakness, instability, or eccentricity, or in one of insane ancestry, they should lead to energetic measures—not only remedial and reconstructive, but to the ultimate regulation of the environment and conduct of the patient in order that more serious development may be prevented.

Spasmodic Wryneck or Mental Torticollis.—G. R. Elliott[2] describes an apparatus for torticollis consisting in a body part with jury-mast extension supplied with a spring which can be adjusted so as to give the necessary amount of twist to oppose the spasmodically acting muscles. The light form of the apparatus seems to meet the indication in cases of mild character. For very intense spasms the so-called Minerva headpiece is devised. This grasps the occiput and cheeks firmly and can exercise any necessary degree of restraining force. [Unfortunately illustrative cases greatly benefited by this apparatus are not detailed. Fixation apparatus tried by numerous observers have usually been unavailing, and become so irksome that they have to be necessarily discarded. The advantage claimed for the present apparatus is that it is so balanced as to exactly balance the amount of spasm in a given case.]

MENTAL DISEASES.

Localization of the Mental Faculties.—C. Phelps[3] makes a study of the localization of the mental faculties, which he is disposed to place principally in the left prefrontal lobe. He says in conclusion: (1) The more absolutely the lesion is limited to the left prefrontal lobe, the more positive and distinctive are the symptoms of mental default; (2) the integrity of the mental faculties remains unimpaired in right frontal lesion, though it involves the destruction of the entire lobe, or even extends to the entire hemisphere; (3) the exceptional instances in which seemingly opposite conditions exist are always reconcilable, on more careful examination, with the assertion of an exclusive control of the mental faculties residing in the prefrontal region of the left side. If, then, the same nature and degree of proof which is deemed sufficient for the localization of other cerebral functions may be accepted in case of the mental faculties, their center of control has been established.

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[1] Phila. Med. Jour., Nov. 22, 1902. [2] N. Y. Med. Jour., Oct. 11, 1902.
[3] Am. Jour. Med. Sci., May, 1902.

Cerebrospinal Fluid in Insanity.—L. Duflos[1] attempts to substantiate the idea that certain mental affections, especially alcoholic insanities, subacute varieties of general paralysis, and senile dementia, may be differentially diagnosticated by examination of the cerebrospinal fluid obtained by spinal puncture. In chronic alcoholic subjects lymphocytes were found in small numbers, but in subacute delirium with hallucinations the proportion of lymphocytes was high, and the author believes that the presence of lymphocytes indicates an inflammatory process, while the presence of polynuclear leukocytes indicates a chronic or subacute process, as in tuberculosis. In paretic dementia lymphocytes were present in abundance, serving to differentiate this disease from the insanities of alcohol and some forms of senile dementia. Babinski and Nagrotte have expressed somewhat similar views.

Insanity.—W. Ford Robertson,[2] before the Section of Psychological Medicine of the British Medical Association, discussed the subject of **toxic action in the pathogenesis of insanity.** He first presented a consideration of the factors upon which normal and morbid mental action depend and called attention to the importance of fault in the cortical neurons, the part played by sensory impulses, the disorders of nerve-cell nutrition, and the conditions arising from deficiency of nutritive material, and, finally, to the effect of toxic action, both direct and indirect, and its result upon nerve-cells. These toxic agents may be (1) exogenous, (2) infections, and (3) autointoxications and autoinfections. In the third group the most important are those arising from the gastrointestinal tract. To this he would attribute a large array of acute and chronic diseases, including several forms of mental disorder. For instance, various forms of arteriosclerosis and its consequences, a large proportion of cases of senile insanity, general paralysis, locomotor ataxia, chronic alcoholic insanity, dementia præcox, idiopathic epilepsy, and most cases of acute and chronic mania and melancholia and chronic Bright's disease. In his conclusion he reiterates that the most important factor in the pathogenesis of insanity is toxic action. His belief is that the great majority of cases of insanity are not primarily diseases of the brain at all, but depend upon toxins elsewhere derived which affect the functions of the cortex by disordering its embolism and often permanently damaging or destroying many of the cells.

F. W. Mott,[3] in opening a discussion on **syphilis as a cause of insanity,** says that syphilis may act in many ways as a cause of insanity: (1) By moral shock the patient may be infected, or believe that he is infected, and the shock may be so great as to lead to insomnia, resulting in melancholia, mania, or delusional insanity. (2) By the effect of the poison and anemia on the general nutrition of the body. It also happens during the secondary stage, when there is usually more or less anemia and leukocytosis with fever commonly present, that the neuropathic or psychopathic individual may become insane. The same condition may arise from the excessive or injudicious use of mercury. Inquiry into the family history of such cases usually determines the presence of hereditary neuropathic or psychopathic factors. (3) The specific poison may cause an inflammation affecting the supporting and nutrifying tissues of the brain, producing meningitis, gummy tumors, endarteritis, and periarteritis. The most severe cases in the author's experience occurred in the first 4 years after infection. (4) Syphilis is a competent cause of arterial degeneration, leading to softening, paralysis, and posthemiplegic dementia. (5) Syphilis plays a very large part in the etiology of general paresis, but the author does not believe that it is absolutely essential to the development of tabetic dementia, though he goes so far in practical work as to say that "where there is no syphilis there is no general paralysis," and asks the very pertinent question whether any one has ever known a case of general paresis to show any of the early lesions of syphilis; whether, in other words, the patient is not immune. And this, too, in view of the fact that paretics in the early stages of their maladies usually expose themselves promiscuously to all sorts of infections.

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[1] Thèse de Paris, 1901.

[2] Brit. Med. Jour., Oct. 26, 1901.

[3] Brit. Med. Jour., Oct. 18, 1902.

Idiocy.—A. Hymanson[1] reports a case of **amaurotic family idiocy.** The child's great-uncle had 11 children; 5 of them died at the age of 2 years. They had large heads, thick faces, and idiotic laugh. They were paralyzed and could not see or grasp anything. The father of the child reported by the author thinks his son's ailment is similar. The child in question appeared to be perfectly normal until the end of the seventh month, when he was able to grasp things and play with them, and with his mother's support could stand. On reaching the eighth month the child ceased to take interest in his surroundings, became listless, apathetic, could not hold up its head, made purposeless movements with its limbs, seemed to be deaf, but was frightened when any one knocked at the door. Sense of taste was not entirely gone, disliked bitter or salt things, but took sweet things readily. During sleep keeps eyes and mouth wide open. Reflexes are present, reaction from faradic current normal. Condition of the child going from bad to worse. Examination of the condition of the eyes by Schapringer showed the peculiar retinal condition which seems to be characteristic of the disease. There was no nystagmus or strabismus. In the posterior pole of each eye could be seen the changes first described by Tay. A large bluish-white spot with softened edges about twice the size of the optic disk, in the center a brownish-red fairly circular dot strongly contrasting with the surrounding patch, suggesting the spot seen in cases of embolism of the central artery of the retina. The optic nerves were atrophic, the optic disks being grayish; the blood-vessels, arteries, and veins were reduced in size. This interesting condition, first made prominent by B. Sachs, has been reported upon more frequently in America than elsewhere; 30 American cases have been noted, 13 in England, 11 in Germany, American cases have been largely among children of Russian and Polish Jews.

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[1] N. Y. Med. Jour., July 12, 1902.

Optic Atrophy.—H. Derby[1] strongly supports the long-time treatment of optic atrophy by the subcutaneous injection of strychnin. He reports a number of cases. The strychnin is preferably injected at the temples. He believes that strychnin is a stimulant to the optic nerve. In certain cases of optic atrophy its subcutaneous use has coincided with an arrest in the progress of the disease and been followed by increase of vision. [The 9 cases reported do not brilliantly support the contentions of the author and are very closely parallel with the experiences of others in the treatment of optic atrophy with strychnin.]

Adolescent Insanity.—Masselon,[2] after reviewing the studies of Krapelin and others, would divide dementia præcox into 4 varieties: (1) A simple form without delusions; (2) a delusional form, also called hebephrenia, marked by excitement intermingled with hallucinations, terror, and confusion of ideas, the symptoms, however, being fugitive and varying at every moment; (3) a catatonic form marked by symptoms of stupor, cataleptic postures, speechlessness or verbal delusions, and incapacity for attention; (4) a paranoid form in which sensory disturbance and delusions develop rapidly with progressive mental enfeeblement. Among the commoner physical signs are dilated sluggish pupils, exaggerated knee-jerks, feeble circulation, and some form of intestinal autointoxication with temporary delirium. The age of onset varies from 17 to 25 years, or, in married women, even to 30 years. The percentage of recovery was but 8 in the delusional, and 13 in the catatonic variety. The causation is obscure, but hereditary taint could often be traced. Detailed accounts of 13 personally observed cases are given

F. X. Dercum[3] presents a very thoughtful and comprehensive article on the subject of **mental disorders in children.** He properly insists upon a distinction between idiocy as a congenital condition and imbecility as, to some extent, an acquired one. Among children delirium, confusion, and stupor are very common, but melancholia, mania, circular forms of mental disturbance, and paranoia are extremely rare, if they appear at all, owing to the lack of mental development, perhaps; but children are occasionally met who exhibit excessive fear, abnormal self-distrust, morbid conscientiousness, even presenting notions of self-blame and making confessions of imaginary sins. There may be present abnormal exaltation and excitement, particularly with a tendency to precocious religious profession. These are especially among children of neurotic heredity. Neurasthenic insanities are not so uncommon in children, but rarely present any well-defined or sharply differentiated forms. They are usually marked by abnormal fears, painful indecision, tyrannical obsessions, irritable impulses, but are incomplete and changeable. Here, too, a neuropathic heredity is practically essential. At the age of puberty, or occurring some years before or after puberty, the dementia præcox of Kræpelin is one of the most common and serious varieties of mental disturbance occurring in the young. It is a dementia which begins insiduously, and is usually progressive. Its first phase is characterized by depression, its second by expansion or exaltation, one phase gradually passing into the next. It is somewhat more common among males than females. Often it is preceded by a long period of listlessness, or the child may even show unusual precocity. There is commonly a neurasthenic element mixed with it. Hallucinations are usual in the first period, and the patient has a tendency to aimless wanderings and sometimes protracted flights from home, for which ordinarily no explanation is made, although occasionally it is stated to be the result of terrifying hallucinations. Speech remains fairly good, although the impaired quality of thought is manifest as soon as the patient talks. Unsystematized or purely systematized delusions may be noted, and in older children comparatively well-systematized delusions have been observed. In the initial period, which lasts for months, and sometimes for a year, the mental attitude is one of continued depression and the delusive beliefs are of a painful character. There is a lack of spontaneity and poverty of ideas; the patient is indifferent to those about him. He may be irritable and show a loss of self-control. After many months, elements of exaltation and expansion gradually make their appearance. Causeless laughter, boisterous conduct, and physical restlessness appear, the patient is declamatory, childish, silly, theatrical, with signs of automatism and a tendency to cataleptic postures. Of those forms of insanity occurring in the adult, probably paresis alone is of importance as an affection in childhood, and it is exceedingly rare. Inherited syphilis is an essential factor, and the disease does not differ in its important features from that observed in the adult, save for the mental symptoms, which are usually those of simple uncomplicated dementia. This is in keeping with other forms of insanity in childhood, showing the absence of those elaborate and well-defined delusions which are dependent upon a mature mental organization.

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[1] Boston M. and S. Jour., May 15, 1902.

[2] Paris Theses, 1902.

[3] Phila. Med. Jour., July 19, 1902.

Melancholia.—Jules Clausse[1] speaks highly of the use of **codein** in certain classes of nervous and mental agitation. The phosphate was the salt employed. Observations were made on 15 patients. He noted that it was particularly beneficial in melancholia with feelings of anguish and moral suffering, often with disturbance of general sensibility. Such patients were rendered cheerful and happy and slept fairly well. The remedy did not constipate the bowels seriously and respiration was improved. He used from 4 to 6 cgm. hypodermically, or from 6 to 8 cgm. by oral administration. Cases with intense excitement and agitation and simple mania were made worse by the remedy.

Under the title "sitophobia" Ruata[2] investigates the condition of 20 insane patients who manifested an **aversion to food,** or sitophobia. In depressed mental states with an anemic brain there was a deficiency or absence of hydrochloric acid. In conditions of mental excitement or agitation the hydrochloric acid was in excess. The author believes the condition of the nervous system affects the stomach, and the abnormal condition of the digestive process in the stomach in turn brings about a

[1] Thèse de Paris, 1902.

[2] Gaz. Med. Lombard., Nov. 10, 1901.

toxic condition of the nervous apparatus. In melancholia an atonic dilation of the stomach is constant, and regular washing out with a soda solution improved its functions. In addition, the author administered hydrochloric acid in these cases.

Puerperal Psychoses.—Meyer,[1] basing his study upon 1104 cases of psychoses in women, found 51 instances of puerperal and lactational insanity; infection being clearly established in 5 of the 51 cases. In 5 mastitis was present, in 3 pulmonary phthisis was observed, in 1 typhoid fever; a hereditary neurotic taint was present in 29, and in the majority of the cases the puerperal or lactational episode was only regarded as an exciting cause. The general conclusion is that there are several forms of puerperal psychoses, but not a single or all-comprehensive form of puerperal insanity. In this group of cases 11 showed melancholia, 4 periodic melancholia, 3 circular insanity of the maniacal-melancholic form, 5 paranoiac states with chronic delusions, 9 acute mental confusion, 4 katatonia, 2 hebephrenia, 2 epilepsy, and 1 hysteria.

Mania.—G. S. Walker[2] reports a case of **sympathetic insanity in twin sisters** which showed some peculiarities. Insanity in twins has been repeatedly observed heretofore. [See YEAR-BOOK, 1902.] In the present instance one sister became acutely maniacal and the second sister, who was sent for, promptly became similarly affected as soon as she saw her twin. They were placed in the same hospital, but in separate wards, and both presented symptoms of violent acute mania. Both improved about the same time and both relapsed at the same time, and finally both again became violent at the same time, and both entered dementia about the same time, this continuing ever since.

Typhoid Psychoses.—Farrar[3] reports cases illustrating various types of psychoses resulting from typhoid fever. (1) He describes initial delirium as the rarest form of psychic disturbance, but presenting the most rapid course and worst prognosis. Over 50 % terminated fatally. This delirium may precede for several days the onset of fever, or the cases may remain afebrile throughout. Such cases often result in the commitment of patients to institutions for the insane. (2) Febrile psychoses often result during the course of the fever and are of the commonest variety. These patients present a good outlook, although the psychic disturbance in 25 % exists for various lengths of time into and after convalescence. (3) There are the asthenic psychoses developed during the period of convalescence, conditions of exhaustion marked by anemia, malnutrition, fever, or the development of psychic disturbance.

Paretic Dementia.—W. Tschisch[4] proposes the following definition for paretic dementia: General paralysis is a parasyphilitic affection of the entire system; the disease depends on a special state of the syphilitic poison (parasyphilitic); the latter decomposes and diffuses its virulence into every tissue of the body. Anatomo-pathologically, as well as clinically, general paralysis is characterized by a proportionate diffusion of the poison in and a destruction thereby of the whole organism; not one organ or tissue is more affected than another during the progress of this disease. The functions of the organs are similarly attacked in equal proportion.

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[1] Berl. klin. Woch., 1901.

[2] Am. Jour. of Insanity, Oct., 1902.

[3] Am. Jour. of Insanity, July, 1902.

[4] Jour. Ment. Pathol., July, 1902.

William Pickett contributes a very important article on the subject of paresis containing a **clinical study of 149 cases** occurring in the Philadelphia Hospital. It is interesting to note that the larger proportion of these cases presented the depressed, nonexpansive variety of paretic dementia. In the diagnosis of the condition the state of the pupils is considered one of the most important elements. The author contends that any abnormality of the pupils not accounted for by local lesions may serve as a sign of paresis. Inequality is the most frequent change, but abnormalities in pupillary reactions are found with almost equal frequency when the examination is minutely conducted The pin-point pupil and the Argyll-Robertson pupil occur an equal number of times, but in some patients the Argyll-Robertson type is more frequently associated in this series with dilated pupil than with a contracted pupil. Of the cases 31 % showed an absent knee-jerk, it was diminished in another 10 %, increased in 49 %, present but normal in 10 %. Epileptiform episodes occurred as the most common accident of the disorder and were the most fatal of this variety of clinical manifestations. Next to it in point of frequency came maniacal outbreaks, then apoplectiform seizures, then stuporous attacks, and finally slight petit mal convulsions. The author contends that an isolated rise of temperature often represents an abortive episode. While recognizing the part played by syphilis, the author contends that hereditary tendency is a strong factor, furnishing a field in which epilepsy produces its malignant work. In 89 cases of the 149, marked heredity was obtainable.

Pellagrous Insanity.—John Warnock[1] reports a number of cases of pellagrous insanity observed by him in the Cairo asylum in Egypt. They usually presented the characteristic rash of pellagra. They presented increased tendon-reflexes, although in 5 instances the knee-jerks were absent and generally a paretic gait was observed in advanced cases. The patient walked with legs well apart but tending to fall after a few feeble steps. Many cases were not even able to stand. In some advanced cases ankle clonus was present, and convulsions were occasionally observed, though epilepsy is so common in Egypt that this may have been a coincidence. General emaciation results in atrophy of all of the muscles of the body, particularly in the last stages of the disease, when loss of control of rectum and bladder is common. Intermittent uncontrollable diarrhea occurs almost invariably. The pale anemic tongue has a smooth slimy appearance at the tip and sides as if stripped of epithelium; the so-called bald tongue of Sandwith is sometimes noted. Mentally the picture is one of melancholia, often with unpleasant delusions of possession of devils, persecution, poisoning, sorcery, impending death, etc. [It is regrettable that the anatomic conditions found postmortem were not set forth in the communication.]

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[1] Am. Jour. Med. Sci., Jan., 1902.

Village Care for the Insane.—Julia C. Lathrop[1] describes the village and family care of the insane as she has seen it abroad in Scotland, Belgium, and France, and believes that under certain restrictions it may be valuable in the management of certain classes of insanity in this country. [It is doubtful if our social systems are compatible with the family and village care of the insane. The lack of constant medical supervision, the undesirable presence of the insane individual in a family, and many other considerations militate against any general adoption of the plan. It would be very difficult, except among those of foreign birth, to find people who could be induced to assume the obligations of the care of such cases. The very people one would select for such a purpose are those who would refuse to undertake it.]

[1] Report of the Twenty-ninth National Conference of Charities and Correction.

CUTANEOUS DISEASES AND SYPHILIS.

BY LOUIS A. DUHRING, M.D., AND MILTON B. HARTZELL, M.D.,

OF PHILADELPHIA.

INFLAMMATIONS.

Erythema Nodosum Complicated with Iritis.—A. W. Allan[1] gives the notes of a farmer's wife aged 33 who developed a typical erythema nodosum on the legs. After the cutaneous inflammation had partly subsided, a fresh crop of nodules appeared, likewise on the legs, with intense pain. The febrile symptoms gradually abated, and about a week later bilateral iritis manifested itself. No synechias formed and the inflammation cleared up in a few days, the erythema on the skin gradually disappearing with the usual discoloration.

The Contagiousness of Erythema Nodosum.—Moussous[2] has observed in the hospital on two different occasions erythema nodosum develop in the patient in the adjoining bed some days after a child had been admitted with this affection. He believes these cases can only be explained by contagion, and concludes that erythema nodosum is not a form of polymorphous erythema, but a specific affection. [This theory, accepted by many, is steadily gaining ground.

Erythema Induratum Scrofulosorum.—A. Whitfield,[3] of London, discusses the nature of this disease, especially with reference to tuberculosis. Bazin, who was the first to describe it, regarded it as scrofulous. Recently Audry excised a nodule and found nothing resembling tubercular structure about it, nor did the animals he inoculated become infected. Whitfield concludes that there are at least two forms of disease, to which the name of Bazin's disease has been given. One of these is essentially indolent, chiefly affecting adolescent girls, and is in some cases undoubtedly caused by the living tubercle bacillus. The other affects women of middle age almost if not quite entirely—usually those who suffer from some form of cardiac weakness—is more acute, more amenable to treatment by rest in bed, and has nothing to do with the tuberculous process. A case allied to it if not identical with this second condition has recently been described by Phillipson under the name of "phlebitis nodularis necrotisans."

Bullous Urticaria.—Malcolm Morris[4] records a case of urticaria occurring in a female infant otherwise healthy, aged 13 months, in which, in addition to urticarial wheals, large bullas were present upon the trunk, limbs, and scalp. Little[1] records the case of an infant 11 days old in whom there was an eruption of bullas upon a generally erythematous skin, seated in the axillas, on the suprapubic region, the backs of the thighs, the cheeks, forehead, and genitalia. At the same time there were elsewhere small urticarial papules.

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[1] Brit. Med. Jour., Oct. 26, 1901.

[2] Arch. de Méd. des Enf., July, 1901.

[3] Am. Jour. Med. Sci., Dec., 1901.

[4] Brit. Jour. of Dermat., May, 1901.

Postdiphtheritic Urticaria.—J. J. Walsh[2] records a case in which, though no antitoxin was employed, urticaria developed at just about that stage in the disease when the eruption would probably have occurred if antitoxin were employed. The author reports the case in view of the fact that objections to the use of diphtheria antitoxin in the public mind is the fact that after its administation an urticarial eruption.occasionally occurs. It is probable that a slight urticaria or an erythema occurs oftener in diphtheria than is generally supposed to be the case.

Thyroid Extract in Urticaria.—J. N. Roussel[3] accidentally found that this drug, which was being employed with the view of relieving obesity and persistent constipation, cured a chronic urticaria with which the patient had been annoyed for several years. Two hundred 5-grain tablets, one thrice a day, were taken. Subsequently the same drug was administered to 3 other patients suffering from urticaria, all of whom recovered, and 2 other cases in the practice of another physician are believed to have been cured.

Diphtheria Antitoxin Eruptions.—Arthur Stanley's[4] observations were made on 500 cases of diphtheria during 2 years' work in a diphtheria ward of a hospital. All cases were treated with antitoxin and the death-rate was 16 %. The virus was prepared at the laboratories of the Royal College of Physicians and Surgeons in London, and was injected in quantities usually of 4000 Behring antitoxin units immediately after admission, but varied from 1000 up to 30,000 units according to the severity of the case. No constant relation between the quantity of antitoxin given and the frequency of cutaneous disorder was noted. Among the 500 cases there occurred antitoxin eruptions in 112 cases; of these 58 were erythemas; 15 erythemas with urticaria; 30 urticaria; 6 scarlatiniform; 3 morbilliform. The erythemas appeared from the fourth to the twenty-ninth day, and urticaria from the fourth to the nineteenth day. The typical diphtheria antitoxin eruption is a marginate erythema in the "psoriasis regions," tending to run into arcs of a circle, lasting about 3 days, and accompanied by slight malaise, and a rise in temperature of about 3° F. The margins of the erythema are raised and turgid, and may be made up of arcs of circles of widely varying diameter according to the phase of spread of the lesions. Slight pigmentation usually succeeds the erythema. The existence of the eruption did not seem to bear upon the prognosis.

An Eruption Resembling Variola Produced by Thyroid Extract.—A. S. Morton[5] states that this drug may, and sometimes does, cause a papular eruption that may be mistaken for the early eruption of variola, especially if any backache or rise of temperature is present. Considerable itching accompanies the eruption and lasts for about 6 weeks after the drug is discontinued, and reappears on resuming the drug.

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[1] Brit. Jour. of Dermat., July, 1901
[2] Phila. Med. Jour., Oct. 12, 1901.
[3] New Orleans M. and S. Jour., April, 1902.
[4] Brit. Med. Jour., Feb. 15, 1902.
[5] Lancet, Jan. 25, 1902.

Electroplating Dermatitis.—Arthur Hall[1] calls attention to this form of inflammation, which is due to the lime-dust employed among the finishers in this trade. The material used is finely crushed lime mixed with a small quantity of olive oil. No moisture of any kind is applied. The lime-dust is unirritating to the skin of the hands provided the epidermis is intact, but if it comes in contact with an abrasion or moist surface a tenacious caustic paste is formed, which tends gradually to form an ulcer unless the lesion is treated.

Poisoning by Fur.—J. C. White[2] calls attention to the occurrence of cases of eczematous dermatitis of the neck and face produced by wearing fur tippets. The type of the efflorescence was papular, distinguished by the flatness and unusual size of the individual lesions, which were both discrete and grouped in confluent areas of moderate extent. The sides of the neck and cheeks are principally affected, and the eyelids have been especially inflamed in several instances. In one or two cases deep, follicular inflammation has followed the first stage of the dermatitis. The tippets in the cases observed were made of the cheaper furs, which are colored by some dark brown dye. In some instances the fur, when wet, stained the skin. The affection yielded readily to the ordinary local remedies for eczema of similar type, but not until the tippet had been laid aside.

Eruptions Due to Orthoform.—W. Dubreuilh[3] states that while it is generally admitted that this drug is usually well tolerated, it is nevertheless sometimes productive of eruptions. When they occur, they are usually (1) erythematous, complicated or not by vesiculation or pustulation; or (2) ulcerative and gangrenous. Two typical instances of the latter variety are given, both in women, one involving the anal and genital region, the other the hands, the lesions being both ulcerative and gangrenous. The author calls attention in particular to the observation, not heretofore noted, that the analgesic property of the drug persists upon the lesions which it itself has produced upon the skin.

Dermatitis Nodularis Necrotica.—L. Tœrœk[4] in 2 cases investigated the seat of the primary focus of the disease, especially with reference to the sweat glands and blood-vessels, and concludes that it starts in the blood-vessels, the first changes in most cases occurring in the veins seated between the cutis and the subcutis, and in the form of a proliferating endophlebitis which is succeeded by an irritative phlebitis. The lumen of the vein is obstructed, a thrombus is formed, followed by necrosis in the neighboring tissues. This leads to necrosis of the skin, the superficial strata of the corium, ulcers and crusts. No microorganisms were found. Dubreuilh also takes this view of the process.

Phlebitis Nodularis Necrotisans.—Philippson[5] under the above title reports a case which exhibited lesions corresponding clinically with the so-called tuberculids of Darier. The patient, a girl, aged 18, two years before coming under Philippson's observation had had an eruption of painful pustules upon the legs, which after a time became ulcers and healed very slowly. When first seen there were a number of rounded ulcers surrounded by a marked halo, irregularly distributed over the legs, the bottoms covered with necrotic tissue. These began as inflammatory spots, which after a week or two became necrotic in the center, forming ulcers which slowly spread peripherally. A few firm, bluish nodules were also observed on the heel and outer side of the foot. Microscopic examination of excised lesions showed that the process was an inflammatory one arising in the blood-vessels, the most marked changes being in the branches of the venous network which lies in the fat-tissue beneath the cutis. The author concludes from the study of this case that the tuberculides begin as a phlebitis.

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[1] Brit. Jour. of Dermat., June, 1901. [2] Boston M. and S. Jour., Mar. 6, 1902.

[3] La Presse Méd., May 18, 1901.

[4] Arch. f. Derm. u. Syph., Bd. LIII, 1901, p. 339.

[5] Arch. f. Derm. u. Syph., Bd. LV, Heft 2.

An Epidemic of Noma.—Blumer and MacFarlane[1] report an epidemic of 16 cases, all occurring in the Albany Orphan Asylum following an epidemic of measles. The mouth alone was affected in 4, the vulva in 2, and the rectum alone in 3 cases. In 5 of these pneumonia was the direct cause of death. The ages of the children ranged from 3 to 12 years. There were nearly twice as many cases of measles among the boys, but no case of noma developed among them. After the cases were isolated no new cases developed. Where the destruction of tissue was not too extensive, thorough cauterization under chloroform with the Paquelin cautery seemed to stop the progress of the gangrene. Of the 16 cases, 9 were examined bacteriologically. One organism was constantly present. It had the form of a leptothrix, having a length of from 10 to 40 times its breadth. The short forms were bent, the long were curly or wavy. As the process continued its course the cover-slips showed a greater mixture of other germs, from which the authors conclude that while originating probably as a simple infection the disease in its later stages is a mixed infection. While it is most frequently due to a thread-like organism of the leptothrix type, it may not always be caused by the same germ.

Disseminated Gangrene of the Skin in Children.—Veillon and Hallé[2] record a case accompanying measles, bloody blebs appearing on the fourth day of the measles, which were soon transformed into ulcer involving the entire thickness of the skin. Bacteriology showed Staphylococcus aureus and an anaerobic bacillus described as Bacillus ramosus; sections of the skin showed the same bacillus.

A Case of Dermatitis Gangrænosa.—Finny[3] reports a case in a boy under 3 years of age. The affection at first appeared to be impetigo. The pustules were scattered over the skin of the suprapubic region, the penis, the wrists, and the back of the hands, were closely set and about as large as a pinhead. At the end of 3 weeks the process had spread to the lower extremities, and at the same time several pustules developed upon the face and scalp. Extensive ulceration set in, which resulted in deeppunched-out, round ulcers covered with pus. Death occurred toward the end of the fourth week. The necropsy revealed general tuberculosis. There was no reason to associate the disease with vaccination or varicella. The presence of tuberculosis has been noted in many of the fatal cases of dermatitis gangrænosa.

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[1] Am. Jour. Med. Sci., Nov., 1901. [2] Ann. de Derm. et de Syph., No. 5, 1901.
[3] Dublin Jour. Med. Sci., Dec., 1901.

ECZEMA, PSORIASIS, LICHEN, HERPES, ACNE.

Vegetative Dermatitis Developing During the Course of Infantile Eczema.—G. W. Wende and H. K. Degroat[1] report 2 cases and refer to others already recorded. In one case an eczema developed at the age of 3 weeks, which in 4 months spread over the entire scalp. During an almost complete remission of the eczema the peculiar lesions about to be described appeared. At the age of 6 months, when the case was observed by the author (Fig. 4), there were 10 well-developed lesions in the scalp, from the size of a pea to a hickory-nut, well elevated, vegetating, and covered with crusts. On the forehead were 2 firm, papillomatous crusted papulo-pustules and vegetations of filbert size. The same kind of lesions but larger existed on the cheeks, one being an inch in diameter and an inch in depth, composed of several coalescent lesions. At two points a small amount of pus could be pressed out, and the most gentle handling resulted in bleeding. On the left leg was an incrusted lesion the size of a

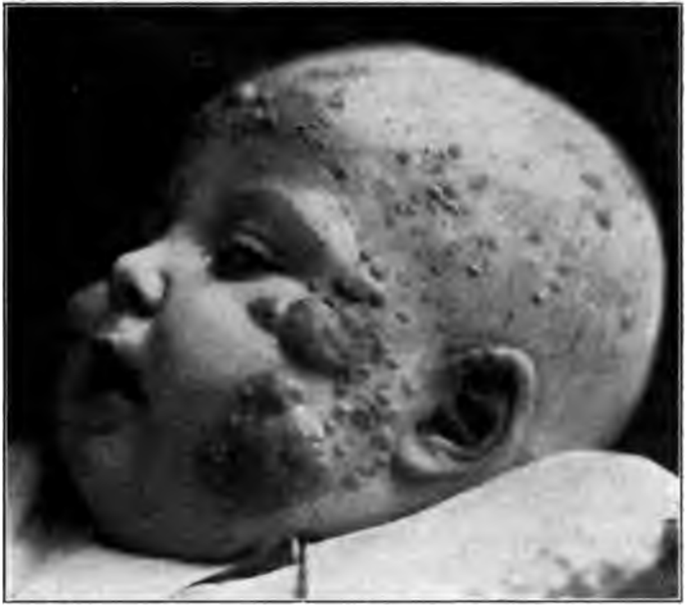

Fig. 4.—Vegetating dermatitis (Wende and Degroat, in Jour. of Cutan. and Gen.-Urin. Dis., Feb., 1902).

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[1] Jour. of Cutan. and Gen.-Urin. Dis., Feb., 1902.

silver dollar. Miliary pustules existed near the larger lesions. Under oleate of mercury ointment in some and sulfur ointment in other places the lesions disappeared, both ointments acting equally well. In 8 weeks the disease was cured. The disease was not regarded by the authors as the result of eczema, but as due to an infection of some kind. Microscopic examination and cultures failed to throw any light on the nature of the process. The cases were similar to 3 cases in nurslings described in 1900 by Perrin. Cases much like these occurring in adults have also been recorded by Hallopeau, under the names "eczema vegetante" and "pyodermite vegetante."

Septic Eczema; Pyogenic Urticaria.—R. Campana[1] reports the case of a patient suffering from urticaria, impetiginous eczema, and a pustule which led to furuncle, all at the same time. The same micrococci were found in all the lesions. There was a true urticaria, and not a mere papular septic dermatitis. In support of the view that this urticaria was of septic origin is the fact that here and there in the infiltrated matters were found blood-corpuscles, some altered and some discolored, and distributed among the epithelial cells were lumps of pigment. This pigment was evidently of corpuscular origin, and was composed of granules, which were all alike and of a relatively small size.

The Origin of Eczema.—Bockhart[2] concludes from his experiments that eczema is an infective inflammation of the skin. Healthy skin may contain within the follicles living but inactive staphylococci, which may take on a more active life by some cause acting from within the body or form on the surface of the skin, which improves the soil in favor of the cocci, this taking place usually in the follicles, augmented metabolism of the cocci causing the excretion of staphylotoxin. As soon as the staphylococcus finds its way from the follicle into the epidermis, through its serotactic influence the formation of papules and vesicles begins. These lesions overlying the follicle then contain serum and cocci, those near by, however, contain serum but almost no cocci. The cocci situated at the opening of the follicle soon increase in numbers, when leukocytes make their way into this vesicle. Later secondary infection of the nearby sterile vesicles take place. Whether the vesicles retain their serous character or become pustules depends on the character of the plasmin of the cocci. The broken vesicles and the moist surface of the skin may become infected by cocci containing an active staphyloplasmin, when such diseases as impetigo and furuncle may complicate the original lesions.

Experimental Researches on the Etiology of Eczema.—Bender, Bockhart, and Gerlach[3] attempt to define the relations between the pyogenetic staphylococci (S. pyogenes aureus et albus) and eczema, distinguishing between the effects of the poisons contained in the living organisms and the effects produced by the toxins elaborated by these organisms. By experiment they found that inoculation of virulent cultures of these staphylococci in agar on irritated human skin never give rise to eczema. Such inoculations, however, produce impetigo, furuncle, and abscess. Filtered bouillon cultures of these staphylococci can produce on the skin acute papular or vesicular eczema, when applied by means of moist warm compresses for one or two days, this occurring upon irritated or sound skin and whether disinfected or not. The primary vesicles of such an eczema are sterile, but later, when the lesions become seropustular, S. pyogenes aureus et albus are found in pure culture. This form of inflammation of the skin runs a similar course to artificial eczema set up by the application of such irritants as turpentine. The staphylococcus toxin, and not the cocci themselves, produces eczema.

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[1] Med. Rec., Mar. 1, 1902, from La Rif. Med.
[2] Monatsh. f. prakt. Dermat., Bd. XXXIII, No. 9.
[3] Monatsh. f. prakt. Dermat., Bd. XXXII, No. 4.

Pyrogallol in the Eczema of Children.—L. Leistikow[1] employs pyrogallol with good results in the eczema of children. An ointment containing from 0.5 % to 2 % of pyrogallol is applied to the affected parts. This strength may be gradually increased to 2 % or 3 % in severe cases. Pyrogallol was always well borne when used in this way. The urine should be watched when this drug is used, although no discoloration occurred in the author's experience. Should an erythematous eruption suddenly appear during treatment, an ointment of zinc oxid or of ichthyol should be applied for a few days.

Eczema Produced by Formalin.—Fisher[2] records his experience on the irritating property of formalin upon the skin. He was accustomed to use this antiseptic in the preparation of museum specimens, especially in the winter, and during several successive winters he suffered from an eczema of the fingers, which was at first attributed to the frequent wetting of the hands, but was subsequently found to be due to the use of formalin. Others who worked with him were similarly affected. Susceptibility to the irritant effects of formalin seems to be more or less gradually acquired. For a time the hands may be daily dipped into solutions varying in strength from 1 % to 5 % without ill effects; then itching and vesicles begin to appear, which result in troublesome eczema if the use of the formalin is not immediately given up. The susceptibility of the skin is much increased after such an attack, mere handling of specimens preserved in formalin being sufficient to cause a return of the disease.

Treatment of Psoriasis by Levico Water.—H. Steiner[3] presented a patient 38 years old, who had had psoriasis for 9 years. He had been treated in different hospitals with tar, pyrogallic acid, arsenic, with but little success. He had constant relapses. He was given a hypodermic injection of half a syringeful of boiled Levico water, in the dorsal region. On the third day a syringeful was injected; on the fifth day, 2 syringefuls; on the tenth day, 3 syringefuls, etc. In all, the patient received 22 injections. There was continuous amelioration, and at the time of presentation the previous diseased skin presented only slight pigmentation. The injections of Levico water are easily made, act rapidly, and gastric disturbance is avoided. In therapeutic action Levico water is very much like the cacodylates. It is claimed that sulfur, which it contains in combination, is an advantage, as it is said to exert a vasoconstrictor influence.

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[1] Amer. Med., April 5, 1902, from Bull. Gén. de Thérap., May 30, 1901.
[2] Brit. Jour. of Dermat., August, 1901.
[3] Med. News, Oct 12, 1901, from Rev. de Thérap., Sept. 1, 1901.

Psoriasis is Seldom Associated with Glycosuria.—Walter Pick[1] investigates this subject and finds that alimentary glycosuria is rarely encountered in psoriasis, the recorded cases, only a few in number, being reported chiefly by German observers. The author thinks that this observation is against the theory of psoriasis being a disease of defective excretion.

Psoriasis and Vaccination.—Weinstein[2] points to the rarity of the occurrence of psoriasis on vaccination cicatrices, and the still greater rarity of the eruption first beginning in that situation. A soldier, aged 22, was vaccinated with calf lymph in two places on the left arm about the middle of October, 1900. The vaccinia pustules were succeeded by red scars. Early in February, 1901, they became covered with white scales, which became more and more marked until they presented all the characters of psoriasis. For a month the two scars were the only parts affected, but the disease then spread to the left elbow, and from thence to other parts of the body. The writer has collected 24 cases in which psoriasis appeared at varying intervals after vaccination. In most of these the patients were healthy, and no history of previous attacks of psoriasis was obtained. Most authorities deny the possibility of psoriasis being inoculated by vaccination, especially if calf lymph is used. Psoriasis is unknown in calves. Vaccination probably acts therefore as a simple injury. The occurrence of traumatic psoriasis after the application of iodin, cantharides, and other chemical irritants, after thermic stimuli, whether of cold or heat, and after a great variety of mechanical lesion is well known. The regions which are typically affected—the elbows and tuberosities of the tibias—are exceedingly exposed and liable to injury. Psoriasis also selects parts habitually exposed to pressure, such as the waist in women, the apex of the scoliotic curvature, or the skin beneath tight garters. It has been known to follow the operation of tattooing, the bite of a horse, and excoriations due to riding.

Parakeratosis Variegata.—T. Colcott Fox and J. M. H. MacLeod[3] report a new case of this rare dermatosis, the first observed in London. The patient was a healthy adult male with a good family and personal history. At the age of 31 a circumscribed red patch, covered with fine scales, appeared in the lumbar region, unattended by subjective symptoms, except cracking on bending. About the same time the palms became thickened and the sense of touch dulled. In 6 months the patient was covered all over except the face with a hard, dry skin. The scalp was scurfy, the hair falling out to some extent, but the nails were healthy. This condition of the skin persisted, in spite of various forms of treatment, for 5 years without change. It consisted of a hyperemic mottling in the form of a network, the threads of which were made up of macules and slightly raised, scaly papules. There was hyperkeratosis of the palms, and, to a less extent, of the soles. The author's conclusions from a study of this case and those reported by other observers are as follows: Parakeratosis variegata is a clinical entity. It attacks chiefly the male sex, usually occurs in adult life, and the patients are generally in robust health when attacked. Its general configuration and histology suggest a vasomotor disturbance associated with edema and infiltration of cells in the corium and secondary changes in the epidermis. The initial lesion is a macule or maculo-papule of small size, flat on the surface, covered with a fine, adherent scale, which may be scratched off without causing bleeding. By their coalescence a peculiar retiform arrangement results, in which areas of normal skin are inclosed, and which, combined with differences in the color of the lesions in the more dependent parts of the body, produces the marbled or variegated appearance of the inflamed skin, hence the name. It affects the skin almost universally, except, as a rule, the face, scalp, palms, and soles. It is subject to remissions and exacerbations, but is peculiarly chronic in its course. Marked subjective symptoms are absent. It is resistant to local treatment. It consists histologically of a superficial inflammation affecting the subepidermal layer, with dilation of vessels, edema, and infiltration of cells, and an edematous condition of the epidermis with more or less defect in the process of cornification. It may be regarded as belonging to a group of superficial inflammations of the corium, with secondary changes in the epidermis, which the authors have provisionally called "resistant maculopapular scaly erythrodermias."

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[1] Berl. klin. Woch., Jan. 20, 1902.
[2] Brit. Med. Jour., May 31, 1902, from Wien. med. Woch., Jan. 25, 1902.
[3] Brit. Jour. of Dermat., Sept., 1901.

Mercury in Lichen Planus.—Lusk[1] reports 17 cases treated with $\frac{1}{12}$ grain of corrosive sublimate 3 times daily, the results being satisfactory. In only 2 of the cases was there a history of syphilis.

Lichen Planus Hypertrophicus.—David Lieberthal[2] discusses the form of disease to which this term is applicable, a number of names having been given to it, such as lichen simplex chronicus circumscriptus, lichen verrucosus, lichen corneus, etc., most of which are appropriate to the process. The diagnosis, etiology, and pathology are considered, and 2 cases (with a photograph) given with the microscopic findings. The question as to the presence of typical elementary lesions can be summarized by stating that in some cases there is a more generalized eruption; in some only a limited number, and in still others none, are present in addition to the warty plaques. We may conclude that the absence of typical elementary lesions does not justify the exclusion of hypertrophic lesions from the disease lichen planus. The elementary lesions will be found at one time or another during the course of the disease. The verrucous form may be due to circulatory disturbance. For those cases in which elementary lesions are also present, and especially more extensive in distribution, the prospect of treatment seems better than for those which present the verrucous formations only. In some cases repeated surgical treatment is called for. Arsenic is more likely to prove useful at the periods when typical elementary lesions are also present.

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[1] Jour. of Cutan. and Gen.-Urin. Dis., May, 1901.

[2] Jour. Am. Med. Assoc., Jan. 11, 1902.

To Prevent Development of Herpes Febrilis.—Agatston[1] says that the following treatment may be employed, but only in the early stage, *i. e.*, when the little vesicles begin to appear: Take a piece of rough linen, saturate a part with pure alcohol, and apply moderate friction to the vesicles until they are converted into little ulcers. Follow this by an application of mercuric chlorid (1 : 1000), allowing it to dry in. If done properly, and at the right time, the little ulcers dry up in a day, without developing into sores.

Herpes Zoster and its Relation to Internal Inflammations and Diseases, Especially of the Serous Membranes.—R. G. Curtin,[2] referring to a former article of his entitled "Is herpes zoster a cause of pleurisy and peritonitis?" brought up the following points concerning those cases: Was the internal disease a zoster eruption of the serous membrane? Was the inflammation of the internal filaments of the nerves communicated to the pleura and peritoneum? Did the internal inflammation cause the zoster, or were both diseases independent and simply coincident? Other cases observed since assist in determining at least two of the questions, and perhaps the third. It may be concluded from the cases cited that inflammation of the serous membrane precedes the zoster; therefore it may be reasoned that the inflammation caused the cutaneous eruption of zoster. These conditions occur too frequently to be simply coincident. A case of zoster is referred to which seemed to be induced by an esophageal carcinoma. Cases of influenza and catarrhal fever complicated with zoster are also referred to, recurring in the practice of the author.

Recent Views of the Origin and Nature of Herpes Zoster.—A. Van Harlingen,[3] after discussing Curtin's views and bringing forward. observations in literature confirmatory of them, takes up the subject of the nature in general of the disease, quoting freely from old and recent articles and monographs. The following conclusions are arrived at: Herpes zoster is a specific, infectious, and possibly contagious exanthem, characterized in its invasion by lassitude, general malaise, chills, increased temperature, and more or less digestive disturbance. Following this in most cases neuralgic pains develop along certain nerve-paths or metameric areas, together with the development of enlarged lymphatic glands. A characteristic exanthem shows itself, which runs through a regular cycle of development, acme, and dicrudescence. In rare cases various visceral complications accompany the affection, such as paralysis of sensory or motor nerves, inflammations of the pleura, peritoneum, articulations, or viscera. The author takes the position that zosteroid eruptions, such as have been observed in poisoning from coal-gas, after the ingestion of arsenic, following injuries of the nerves, moral shock, hysteria, and probably other conditions, are to be distinguished from "true herpes zoster" as defined above.

Skin Eruptions in Malaria.—David Riesman[4] cites the diseases of the skin in which malaria has been believed to have been a factor, and from his own experience draws the following conclusions: Skin eruptions are not rare in malarial infection, the most frequent being herpes and urticaria, neither presenting any specific characters. Both may occur in any stage of the malarial paroxysm, although urticaria is most frequent in the febrile, and herpes in the sweating stage. In obscure cases herpes and urticaria, especially the former, may have considerable diagnostic value. Three types of urticaria are recognizable: that accompanying the paroxysm, usually the febrile stage; that taking the place of the chill; and that substituting the entire paroxysm. In their appearances these do not differ among themselves, nor from urticaria due to other causes. In cases of urticaria of obscure etiology, the blood should be examined for plasmodia. Whether found or not, quinine is worthy of a trial.

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[1] N. Y. Med. Jour., Dec. 14, 1901.

[2] Am. Jour. Med. Sci., Feb., 1902.

[3] Amer. Jour. Med. Sci., Jan., 1902.

[4] Amer. Med., Mar. 22, 1902.

Traumatic Zona.—R. Millon[1] describes 2 cases. One was that of a girl aged 11, who struck a table while running, the blow being on the anterior part of the thorax. Three days later a crop of herpetic vesicles appeared, which in a few days spread from the sternum to the spinal column. The usual burning and pricking sensations were present, but there was no fever nor general disturbance. The second case, occurring in a girl of 13, was in most respects much like the first case, the eruption appearing in the region of the blow, occasioned by a fall on the arm.

Impetigo Herpetiformis in a Man.—Gunselt[2] records an instance of this rare disease occurring in a man. This case, together with a few other cases, goes to show that the disease is confined neither to pregnancy nor to the female sex, as was at one time stated.

Acne and Sycosis Treated by Exposures to the Röntgen Rays.—W. A. Pusey[3] records a number of cases in which this therapeutic agent proved very useful. The author states that there is no other affection which he has undertaken to treat with Röntgen rays that has proved so tractable as acne. The favorable results have been produced more quickly and with less effect upon the tissues than he should have imagined was possible when he first undertook the treatment of the cases. He uses a very weak light, employing a fairly soft tube and as weak a current as would illuminate the tube. The treatment is stopped at the first sign of pigmentation or erythema, and in no case has improvement failed to appear simultaneously with the development of these evidences of the rays upon the tissues. The susceptibility of these subjects to the rays was so marked that he sounds a note of warning against the use of anything but the weakest light, one that is just sufficient to show as a faint green glow in the tube.

Intradermic Injections in Acne Rosacea.—D. H. S. Tuthill[3] describes the case of a man aged 34 who had been affected with acne rosacea for 17 years. The disease involved the nose and extended out on the cheek, and was very disfiguring. A solution of pepsin in distilled water was injected beneath the reddened area. Each treatment was followed by considerable swelling, which would subside by the following day. In all, 5 injections were made, covering a period of about 3 weeks. In a month from the first treatment the acne had vanished, but the reddish color of the original area was still apparent. Active exfoliation of the skin followed the injections. At the end of 2½ months not a sign of the original disease remained.

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[1] Méd. des Enf., July, 1901. [2] Arch. f. Derm. u. Syph., Bd. cv, Heft 3, 1901.
[3] Jour. Cutan. and Gen.-Urin. Dis., May, 1902.
[4] Medicine, Oct., 1901, from Merck's Archives, July, 1901.

Folliculitis (Acne) Exulcerans Serpiginosa Nasi (Kaposi).—E. Finger[1] reviews the literature relating to the 5 cases of this rare cutaneous affection hitherto reported, and adds another case. The first manifestation was a small papule, which appeared on the tip of the nose and rapidly ulcerated, apparently as the result of scratching. It was regarded as syphilitic and for 3 months was treated accordingly, but the lesion increased rapidly in extent. When first seen, the ulceration completely surrounded the right nasal orifice and involved the corresponding ala nasi and the contiguous portions of the septum and upper lip, and also the mucous membrane of the right nares, to a distance of about 1 cm. from its orifice. The ulcerated area was surrounded by an inflammatory areola, and was covered by a yellowish, thick exudate, under which there were coarse granulations. An ordinary acne eruption, with numerous comedones, was more or less abundant upon the face, chest, and back. Local treatment only was used, consisting of 2 % borovaselin. At the end of 3 weeks the ulcerated area was gradually healing from without inward, but on the neighboring sound skin several new ulcerating papules had appeared. Five per cent. borovaselin was ordered, and, in addition, the ulcerated surfaces painted daily with a 2 % solution of carbolic acid. In 1 month all the lesions had entirely disappeared, the ulcerated surfaces showing cicatricial tissue. Microscopic examination showed that the process was of a simple inflammatory nature, follicular in origin. Bacteriologic examination showed numerous microorganisms, especially staphylococci. No tubercle bacilli were found. The case adds to the evidence which goes to show that occasionally there occur serpiginous ulcerative cutaneous lesions of follicular origin, which are neither syphilitic nor tubercular.

PARASITIC DISEASES.

The Tricophyton of Human and of Animal Origin.—Jeanselme,[2] in an article on tricophytosis, says that the disease, when of human origin, does not leave a scar, while the variety of animal origin is prone to leave a mark. If a case of tricophytosis, on the scalp or beard, suppurates spontaneously, we are justified in suspecting that the disease is of animal origin and that infection occurred from a horse. The pyogenic character of animal tricophytosis is not due to microbes such as the stapyhlococcus. Microscopic examination of the pus shows leukocytes and spores, but no pus germs; nor are colonies of bacteria obtained from the pus, but only cultures of tricophyton. This pyogenic power is the most important characteristic of animal tricophyton and aids us in distinguishing it from the human variety.

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[1] Med. Rec., May 3, 1902, from Wien. klin. Woch., Mar. 6, 1902.

[2] Internat. Med. Mag., May, 1902, from Jour. de Méd. Inter., Jan. 1, 1902.

Ringworms from Canaries.—MacLeod[1] presented a photograph of a case of ringworm in a boy, aged 9, of interest owing to the unsuual source of the infection. There was a well-defined plaque upon the back of the left wrist about 2 inches in diameter, reddish in color, uniformly elevated above the surrounding skin, with a slightly raised vesicular border. The patch, which had lasted about 10 days, was peculiar in that it had not cleared up in the center. The disease was traced to a pair of pet canaries which the patient was accustomed to feed upon his hand. The skin of the head and neck of both birds was bald, with only stumps of feathers, but was not inflamed. The stumps of feathers showed spores and fragments of mycelium. The spores were irregular in shape and size, and seemed to be less resistant than those of ordinary ringworm, breaking up readily in liquor potassæ. Attempts at cultivation of the fungus were unsuccessful.

Ringworm.—B. Squire,[2] in considering the subject of special schools for children affected with ringworm, advocated by some, states that his long experience in the treatment of this disease would go to prove that this is unnecessary and inexpedient, and that he has found that with ordinary precaution children so affected can be brought up with unaffected children without much danger of contagion.

Treatment of Ringworm of the Scalp.—W. A. Jamieson[3] reviews the various methods of treatment in vogue, and concludes by recommending that the scalp be kept entirely clear of hair by close cutting or shaving, and then kept perfectly clean by a fluid superfatted potash soap, applied twice daily, the surface of the scalp being kept soft, polished, and adapted for the reception of the parasiticide, the most useful one being composed of precipitated sulfur 1 dram; salicylic acid, B-naphthol, and ammoniated mercury, each 10 grains, and lanolin 1 ounce, to be rubbed in for 10 minutes twice daily.

Goosegrease as an Excipient in Ringworm of Scalp.—G. T. Jackson[4] refers to the value of this substance as an excipient in the treatment of tinea trichophytina tonsurans. There is much goosegrease in the market that is not genuine, the best quality being very costly, for the reason that it is the fat taken from the dead but uncooked goose, a few ounces of fat costing as much as the whole goose. That employed by the author is made by the Schiefflin Company. For tinea tonsurans an ointment is made of ʒss to ʒj of the crystals of iodin to ℥j of goosegrease, to be worked into diseased areas by means of a stencil or stiff paint brush. It is also useful in tinea sycosis.

Pinta, a Tropical Disease.—Osborne Browne[5] says that Edgar and Buchanan[6] both allude to pinta as a disease of tropical America. Browne found it very common on the Gold Coast, where the natives erroneously attribute it to yaws, although the latter disease may have occurred years before the pinta. At first it begins as a very itchy dark bluish or black spot, as a rule on the back of the hand or dorsum of the foot; although it not so unrarely extends to the palms or soles. When the disease has gone on for a considerable time, it loses its initial dark color, at all events in places, and is succeeded by a grayish-pink or white color, denoting a loss of the natural pigment of the skin. It is said by natives to extend into Hausaland about Kano, where it is called "tangere." The fungus is in all cases identical. Natives, may, moreover, also lose the pigment from their skin by a burn, wound, or old parasitic diseases (including pinta) long since died out.

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[1] Brit. Jour. of Dermat., May, 1901. [2] Brit. Med. Jour., Feb. 8, 1902.

[3] Edinb. Med. Jour., vol. VII, 1900.

[4] Jour. of Cutan. and Gen.-Urin. Dis., June, 1901.

[5] N. Y. Med. Jour., July 20, 1901, from Jour. of Trop. Med., June 15.

[6] Jour. of Trop. Med., April 15, 1901.

Dermatomycoses in their Relation to Allen's Iodin Test.—Jacob Sobel[1] concludes from his observations that this test will serve to bring into greater prominence visible parasitic lesions, and into view pale, hidden, and sometimes invisible ones. A positive reaction to Lugol's solution in the form of a deep mahogany or dark brown discoloration is strong presumptive evidence that the lesion is parasitic. In the absence of absolute proof of the parasitic nature it is of the greatest aid, and in the presence of a parasitic skin lesion it serves to make assurance doubly sure. It is, however, not of service in differentiating one parasitic disease from another. [The test consists in applying to the diseased areas, or to suspected ones, a solution of iodin, preferably Lugol's solution (iodi., 5; potass. iodi., 10; aqua, 100). In the diseased areas an intense dark brown or deep mahogany discoloration takes place at once, which shows itself in marked contrast to the surrounding healthy tissues, which latter stains a very light yellowish color.]

Cutaneous Blastomycosis.—F. H. Montgomery[2] gives a summary of the clinical, pathologic, and bacteriologic features of this disease, known also as blastomycetic dermatitis, the latter name being given to it by Gilchrist. The description of the disease is based upon a study of 14 cases, under observation for a period of from 4 months to 4 years. The histopathology was studied in 13 and cultures obtained in 11 cases. The disease, briefly described, bears a close resemblance in its clinical features to verrucous tuberculosis of the skin, lupus vulgaris, and carcinoma. The histologic structure also bears considerable resemblance to those diseases, yet differs distinctly from them. Under the microscope sections show a very uneven surface marked by irregular projections and depressions, the whole covered more or less with irregular masses of broken-down material composed of pus, blood, epithelial cells, and various bacteria. The corneous layer varies greatly in thickness, in some cases extending in thickened masses deep into the corium between the distorted papillas. The striking and characteristic changes occur in the mucous layer, which is the seat of extensive hyperplasia, sending down processes deep into the corium, the latter containing the miliary abscesses which are peculiar to this disease. The organisms characteristic of the disease are found in the miliary abscesses both of the epithelium and of the corium.

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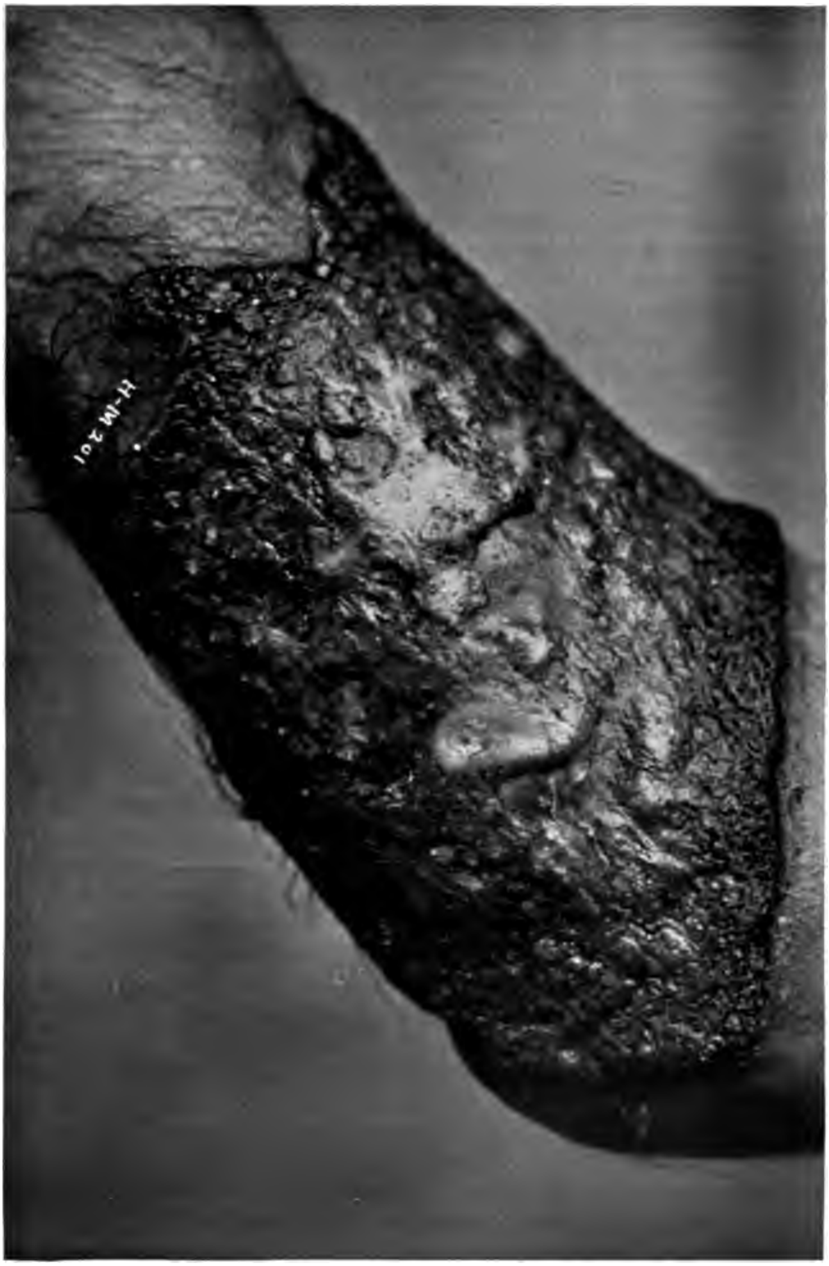
[1] Jour. Am. Med. Assoc., Mar. 15, 1902.

[2] Jour. Am. Med. Assoc., June 7, 1902.

PLATE 3.



and are always surrounded by more or less evidence of inflammation. Giant-cells frequently contain one or more of the parasites, the number of the latter present varying greatly in different cases. A good section will show from 2 to 6 organisms; sometimes they are more abundant; other sections will show none. Distinct budding forms can be found in most cases readily. The diameter of the organism varies from 70 to 20 microns. They appear as double-contoured highly refractive bodies, in some of which granular contents, vacuoles, or shining spore-like bodies can be distinguished. In cultures the organism appears as a mycelium-forming or budding fungus, of which there are probably several varieties. Practically all cases of the disease in which potassium iodid has been administered internally have improved decidedly, but only a small percentage of the cases have recovered entirely under that treatment alone. Cutaneous blastomycosis and protozoic dermatitis are undoubtedly closely related diseases, if not varieties of the same process. (Plate 3.)

Blastomycetic Dermatitis.—Hektoen[1] records a case of dermatitis caused by a blastomyces. The patient was a man aged 50, whose affection dated from 4 years previously, when a red spot appeared on his leg and gradually extended; this was removed by operation. A year before he was seen by Hektoen a warty growth appeared on the hand. Sections of the affected part showed blastomycetes; these were also grown in cultures forming mycelia. The described organisms differ in many points from those described by Gilchrist and Stokes,[2] but the histologic changes caused by them were similar. Rats and mice subcutaneously inoculated died in 5 days and the organisms were regained from the point of inoculation, but not from the internal organs; a dog experimentally used proved refractory to the organism.

Blastomycetic Dermatitis.—Two cases are reported by F. J. Shepherd[3] successfully treated by potassium iodid. The patients were males aged 38 and 72. The lesions in each case were on the face. Diagnosis was confirmed by finding the budding yeast forms in the tissues. Curettage and cauterization of the affected surface were without result, but improvement was noted in a week after beginning the iodid treatment, the dosage varying from grs. 20 to 60 t.i.d. (See Plate 4, Fig. 1.) Another case is reported by Walker and Montgomery[4] which is of interest in being the only instance recorded in which an undoubted cutaneous blastomycosis has been followed by a systemic infection with blastomycetes, 7 years after the beginning of the lesion on the back and face. Death was diagnosed as due to miliary tuberculosis secondary to the cutaneous lesion. Microscopic examination of the lung tissue, however, showed the characteristic organisms which seemed to have caused inflammatory changes comparable to those found in pulmonary tuberculosis. During the progress of the disease no remedy had been of any benefit.

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[1] Brit. Med. Jour., Dec. 28, 1901, from Jour. of Exper. Méd., vol. III, No. 26.
[2] Johns Hopkins Hosp. Rep., vol. I.
[3] Med. News, April 26, 1902, from Jour. Cutan. and Gen.-Urin. Dis., April, 1902.
[4] Jour. Am. Med. Assoc., April 5, 1902.

The Immediate Diagnosis of Blastomycetic Dermatitis.—A. W. Brayton[1] refers to 2 cases of this disease, and calls attention to the point that the microscope for the immediate diagnosis is one of our greatest aids. Dr. Dodds, of Indianapolis, who examined the histology of these cases states that a quick method for the demonstration of the yeast plant in the tissue is as follows: Secure a small fragment of tissue from the advancing margins, and place this upon a glass slide and wash in ether 2 to 5 minutes. Macerate in a 20 % to 30 % potassium hydrate solution for 5 to 10 minutes, or until the blood-cells and extraneous bacteria are destroyed. Examine the specimen with a $\frac{1}{8}$ to $\frac{1}{12}$ objective. Pus taken from a small abscess and treated in a like manner will answer the same purpose as the tissue itself. The potash solution has no action on the yeast cells, and they stand out in the macerated material as doubly contoured, highly refractive bodies from 5 to 20 microns in diameter. They are almost always in the budding stage, and it is not uncommon to find several organisms in the same field.

Oidiomycosis of the Skin and Its Fungi.—H. T. Ricketts's[2] valuable contribution to this new subject is a lengthy and well-elaborated article in the form of a book, a summary of which may be given as follows: The history and pathogenic properties of oidia; a summary of so-called blastomycetic infections in man; clinical, mycologic, and histologic study of 12 cases of this disease; and a critical analysis of blastomycetic infection of the skin, constitute the basis of the work before us, which is made complete by a bibliography, a series of drawings in color illustrating the fungus and the histologic structures. The author finds that there is, essentially, a uniform clinical history, which is briefly as follows: (1) An incipient papule which becomes pustular, yielding a glairy, somewhat tenacious pus; (2) gradual extension of the ulcerating surface, which is soon covered with coarse, soft, and friable papillas, and is surrounded by a reddened areola in which many minute abscesses are visible; (3) cicatricial healing in the oldest portions of the lesion as the border advances; (4) a variable amount of pain, depending on the site involved, upon a temporary increase or decrease of the virulence of the specific infection, and the amount of secondary infection present; (5) the absence of lymphatic glandular involvement in a great majority of cases; (6) the protracted and progressive course of the disease, which may extend over 10 or 12 years, and in the face may cause great cicatricial deformity; (7) periods of rapid extension, interrupted by periods of relative quietude; (8) the tendency of the scar to eventually approach the appearance of normal skin; (9) the upper extremities (hand and forearm) and the face are most frequently attacked, although no region is exempt; (10) extension to mucous surfaces does not occur readily; (11) the absence of general toxic disturbances, attributable to the local infection; (12) the gross anatomy is characteristic and uniform in all essentials. In addition to the features mentioned in paragraphs 1, 2, and 3, naked-eye inspection of a cross-section shows, from without inward: (*a*) a papillary zone, composed of

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[1] Jour. Am. Med. Assoc., Feb. 1, 1902.

[2] Jour. of Med. Research, vol. vi, No. 3, Dec., 1901.

PLATE 4.

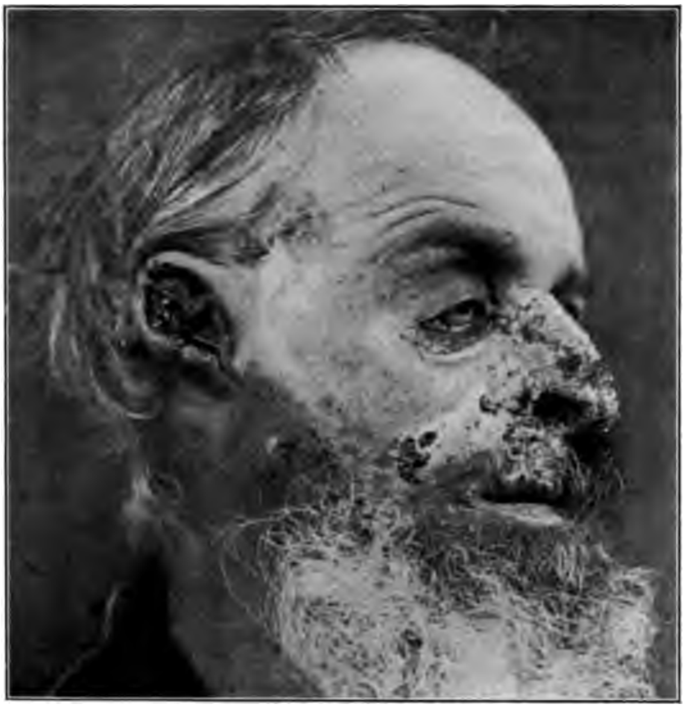

Fig. 1.—Shepherd's case of blastomycetic dermatitis (Jour. of Cutan. and Gen.-Urin. Dis., April, 1902).

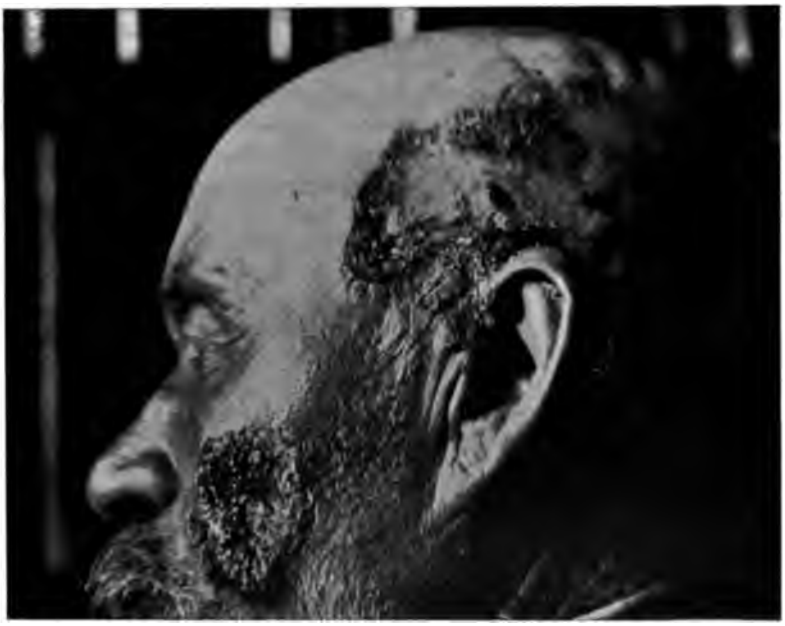

Fig. 2.—Rickett's case of oidiomycosis (blastomycosis) of the skin (Jour. of Med. Research, Dec., 1901).

a superficial layer of isolated villiform processes, and a deeper layer of similar processes which are united side by side; (*b*) a homogeneous, vascularized, grayish-red, cellular zone, in which are formed minute abscesses; (*c*) an unaltered layer of subcutaneous fat, as the limit of deep extension. The following conclusions may be given: The so-called protozoic disease of Posadas, Wernicke, and others; Busse's and Curtis's saccharomycosis hominis; and Gilchrist's blastomycetic dermatitis, are all various manifestations of the same disease. The condition in the skin possesses constant clinical and histologic characteristics, which separate it positively from all other skin diseases, particularly verrucous tuberculosis, carcinoma, and syphilis. The organisms isolated from various cases differ in minor respects among themselves, but are so closely related morphologically and histologically as to justify their inclusion in a common genus, *Oïdium;* they are thus analogous in a pathogenetic sense to the fungi which cause actinomycosis, and to those causing trichophytosis. The variations among the organisms allow the recognition of 3 morphologic types: namely, blastomycetoid or yeast-like, oidium-like, and hyphomycetoid. There are 2 histologic forms of the disease in the skin, the eosinophilous and the noneosinophilous, the former being associated with the mold type of the organism. Oidiomycosis is an appropriate name for conditions caused by the organisms, and oidiomycosis cutis for the disease as it occurs in the skin. Aside from the infections considered in this communication, certain cases which have been described in the literature from time to time indicate that oidium-like organisms may cause other severe pathologic conditions in man. (Plate 4, Fig. 2.)

Pigmentation of the Skin Caused by the Demodex Folliculorum.—Dubreuilh[1] calls attention to the fact that the demodex, while usually a harmless occupant of the follicles, when it exists in great number may give rise to abnormal pigmentation, as shown in the following case: A woman, aged 40, brunet, had noticed for several years a fawn-colored discoloration around the mouth extending along the border of the jaws from one angle to the other. This discoloration was not accompanied by itching or inflammation. Upon close inspection the skin was found to be slightly wrinkled and roughened. Similar but less marked pigmentation existed upon the lateral and anterior parts of the neck and upon the chest. Upon examining scrapings from the affected parts with the microscope the demodex was found in extraordinary abundance. The author refers to similar cases observed by Amicis and Majocchi.

Blue Pigmentation Following Injections of Morphin.—Thibierge[2] reports the case of a woman, aged 40, addicted to the use of morphin from the age of 16, in whom pale, bluish spots, similar to those produced by tattooing with India-ink, symmetrically distributed over the thigh, had followed injections of morphin. These spots were rounded or elongated, 2 to 3 mm. in diameter, with a slight cicatricial depression. Histologic examination of the spots showed the presence of black grains of unequal size, insoluble in alcohol, potash, and concentrated acids, and not giving the reaction for iron, and others transparent and refractive, with sharp corners. The first seemed to be particles of carbon; the others, particles of silica.

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[1] Jour. de Méd. de Bord., 1901, No. 4.

[2] Ann. de Derm. et de Syph., Nos. 8 and 9, 1901.

Permanently Cured Case of Ichthyosis.—Bockhart[1] records an instance of a girl aged 8, who for 9 years was rubbed with sulfur ointment 3 times a day. On account of the odor, she was educated at home. At the date of the report the patient had attained womanhood and had been free of the disease for 6 years. The entire surface except the head, face, joints, palms, and soles was affected. A daily bath was given followed by inunctions with a 5 % sulfur ointment.

Scleroderma.—Neumann[2] describes 5 cases; in 2 there was goiter, in 4 the disease began with rheumatoid pains, the diagnosis of chronic rheumatism having been made in the beginning. Attention is called to the likeness between extensive scleroderma and Addison's disease. The prognosis he considers grave; notwithstanding the reports in literature of stated cures. Little benefit in the cases cited was obtained from internal medication, but in 3 of the cases marked improvement followed the persistent use of hot-air treatment.

Etiology of Sclerema of the Newborn.—Comba[3] gives the notes of 2 cases. One infant was born at term, fairly well developed, but died, the autopsy showing generalized infection with the bacillus of Friedländer which had taken place through the umbilicus. In the other case the same bacillus was found associated with the streptococcus, infection probably occurring through the respiratory mucous membrane. The author thinks that in all cases there is a septicemia. The microorganisms or their toxins produce such a disturbance of centers that the temperature falls, and the induration of the subcutaneous adipose tissue follows. He does not regard the disease as a special nosologic type, but rather as a result of nephritis and of circulatory disturbances secondary to an infectious process.

Elephantiasis.—J. M. Bertolet[4] records an unusual form of this disease occurring in a woman who had never been outside the State of Pennsylvania. The legs and feet were enlarged and deformed in a singular manner, the feet spreading out laterally, forming wide feet. Vesicles, blebs, and great itching were notable symptoms. An examination of the blood disclosed living filarias. This find was subsequently confirmed by J. C. DaCosta, Jr. The patient died in great agony. The autopsy revealed the lymphatics clogged and dilated enormously, and that gangrene had attacked the right foot.

Etiology of Alopecia.—D. L. Parker[5] advances the theory that alopecia is due to autoinfection arising from absorption by the blood of some noxious substance emanating from the air-cells of the lungs, where it has been elaborated by decomposition of the organic matter which exists in respired air. A distinction is made between costal and abdominal breathing as it usually occurs in women and men as influencing baldness. The author has observed that irregular breathing is common in the bald, and that tuberculous persons are seldom subject to baldness. To demonstrate the theory advocated a series of elaborate experiments with birds and animals was conducted, which seemed to justify the conclusion that "the results of the experiments show that when respired air from the human lungs is kept chambered long enough for decomposition of its organic matter to take place, there is developed a substance which, when introduced into the blood of certain animals, exerts a selective poisonous action upon the hair, or tissues analogous to hair, with which such animals may be endowed, and beyond this, so far as careful observation can determine, it produces no effect whatever." "Trichotoxicon" is the name given by the author to the substance that is noxious enough to produce alopecia. [The apparatus used and numerous photographs of the birds and animals used in the experiments illustrate the article.]

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[1] Monatsh. f. prakt. Dermat., Dec. 15, 1901.
[2] Deut. Arch. f. klin. Med., Bd. LXX, 1901.
[3] La Clinica Med. Ital., May, 1901.
[4] Amer. Med., Nov. 9, 1901.
[5] Med. Rec., July 13, 1901.

Alopecia.—Balzer[1] advises for the treatment of this disease the application of a lotion made of $\frac{1}{5}$ part corrosive sublimate, 1 part acetic acid, 100 parts alcohol, and 50 parts each of alcoholic solution of lavender. After drying, the scalp is rubbed with 30 % lactic acid. Ohmann-Dumesnil[2] recommends the use of a current of static electricity applied for 5 minutes daily. A felt cap is placed on the head and a brass ball electrode is passed over the cap, thus stimulating the scalp. The positive electrode should be applied to the scalp and the negative held in the hand. Another method, equally good if not better, consists in the use of stabile cathodal galvanization, a large sponge electrode moistened with salt water being connected with the negative pole, and a current, gradually increased to a strength of 6 milliampères, is applied until the scalp turns reddish.

Is Alopecia Areata Contagious?—Cruyl[3] was unable to trace any contagion in 30 cases observed by him. He does not think that the existence of this disease in epidemic form has been proved, notwithstanding the reported instances in which it has occurred in barracks and institutions. [The disease would seem to be much commoner in Belgium than in this country.]

Alopecia Areata; a Clinical and Experimental Study.—N. Walker and M. M Rockwell[4] examined 63 cases of this disease, with the view of determining either the parasitic or neurotic origin of the disease, the hairs being examined microscopically. In 2 cases portions of skin were excised and examined, in both cases of which "the organisms were found in the follicles." The conclusion is reached that the nervous element in this disease has been too much insisted on by authors. Several histories are given illustrating direct contagion. Organisms were found upon the hairs in all the cases examined, some of the hairs being surrounded by bacteria much as in the case of ordinary ringworm with fungus.

Remarkably Sudden and Complete Loss of Beard and Hair of Scalp Followed by Improved Vision.—J. R. Clarke,[5] of Marion, Kentucky, reports the case of a man aged 86, of fine constitution, and with a history of almost uninterrupted good health until 10 years ago, when he was attacked with hemoptysis, which continued frequently until 1 year ago; he was often expected to die from exhaustion following the great loss of blood, but at no time were there any pyrexic symptoms nor complications. He arose one morning feeling as well as usual, and after washing his face and combing his hair, ate breakfast, went into the sitting-room, began reading, and became so interested that when his wife asked him to get ready for dinner he was much surprised, as he did not think it possible for the time to pass so rapidly. He went to the mirror to comb his hair and beard, the latter being thick and long, covering the upper portion of his chest. Finding the beard somewhat tangled, he parted it with his fingers. He dined as usual, not thinking of the beard, until his wife inquired what ailed his beard; he replied, "Nothing," but on grasping it in his hand, to his astonishment it parted without sensation from his face, and he held it in his hand. Going to the mirror he found that the hair was everywhere else loose and falling, including the scalp, and by simply rubbing with the hand it all suddenly and easily came out. In a few days all the hair had disappeared from the entire surface of the body. A short time after losing the hair, the eyesight began to improve very remarkably, there was no more hemoptysis, and the general health also improved in a marked degree.

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[1] Jour. des Praticiens, Aug. 24, 1901.
[2] Amer. Med., April 15, 1902.
[3] La Clinique, April 27, 1901.
[4] Scottish M. and S. Jour., xi, 1901, p. 12.
[5] Amer. Practitioner and News, July 15, 1901.

Five Cases of Moniliform Hair Associated with Alopecia Areata.—David Walsh[1] records 5 cases in which hairs were found of moniliform type, although differing from the sharply defined condition of monilithrix. The latter is congenital, and runs in families, while those affected have the hair of the head short and scanty, so that they are more or less bald. The eyebrows and any part of the hairy body may be affected. The description of monilithrix just given differs widely from that noted in the cases here reported, in which the hair, although alopecia patches were present, was not short and scanty. The peculiarity was found only in hairs that had turned or were turning white. It did not involve hairs on other parts of the body. The nodes in most of the cases were of irregular length, and the pigment had no distinct relation to the internodes, which is unlike the condition in monilithrix, in which the pigment is found collected in the internodes.

Crude Petroleum of No Value in Baldness.—G. T. Jackson[2] has made many and prolonged experiments with this remedy in the various forms of baldness, some of them extending over a period of from 3 to 7 months, the results being unsatisfactory.

Treatment of Alopecia Areata.—B. Squire[3] describes an ordinary case of this disease occurring in a man aged 37, the face as well as the scalp being affected. An ointment of red iodid of mercury, gr. xxx to the ounce of petrolatum, was applied for a period of 6 months, about one-eighth area of the scalp being treated each day. The hair was retained in patches, some being 3 inches long, others downy, but the eyelashes and eyebrows became complete.

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[1] Brit. Med. Jour., April 12, 1902.
[2] Jour. of Cutan. and Gen.-Urin. Dis., June, 1901.
[3] Brit. Med. Jour., April 12, 1902.

Treatment of Alopecia Areata by X-rays.—Holzknecht[1] reports the case of a successful cure of this disease in 4 months by x-rays. The patient, a male aged 18, had suffered from several patches for 5 months. As a control experiment one patch was not treated by the rays, but an area covering the other patches, together with the healthy scalp, was exposed for a total period of 2 hours, made up of frequent short sittings. A week after exposure the hair fell off all the exposed parts. In 3 weeks the alopecia areas became red; the normal areas were bald but not red. In another month there was a growth of new hair on the alopecia areas, while the rest of the scalp remained bald. The condition was thus the reverse of that at the beginning of treatment. After 4 months' treatment the whole area exposed was covered with hair; the untreated patch, however, was still bald. This was afterward cured. The author does not consider the effect due to bactericidal action, as he finds that exposure of bacteria to the strongest rays at a focal 15 cm. for an hour only hinders their growth for a time. The same exposure to the scalp would cause severe ulceration.

Superfluous Hairs and the Röntgen Rays.—N. T. Schofield[2] states that his experience is adverse to the employment of the Röntgen rays for the permanent removal of hair. In a case in which the hairs fell out they reappeared in a month thicker and stronger than before, and the skin was thickened and hardened. A few more applications were made, but without definite result. The case of an assistant at a hospital whose hands were scarred and had a thick growth of hair on them from overexposure to the rays is referred to.

James Startin's[3] experience goes to show that the Röntgen rays, if carefully employed in exposures of 10 minutes on consecutive days for about 2 weeks at intervals of from 2 to 3 months, will generally effect a complete removal of hair.

David Walsh[4] has not found the use of the Röntgen rays as a depilatory so satisfactory as Schiff has found it. Exposure to the rays from 10 to 15 minutes, with a tube a few inches from the skin, caused the hair to fall out in about 10 days, but in a few months the growth returned. The author has practised successfully a combination of the x-rays and electrolysis. After the hair has fallen out from the rays, the needle is placed in the follicle and allowed to remain there for 20 or 30 seconds. Resolution and patience are requisite on the part of the person to be operated on.

The Röntgen Rays in Diseases of the Hair.—Ed. Schiff[5] has obtained good results in the treatment of, among other diseases, hypertrichosis, sycosis, favus, tinea tonsurans, folliculitis, furunculosis, acne, lupus vulgaris and erythematodes, psoriasis, nevus, and telangiectasis.

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[1] Brit. Med. Jour., Feb. 22, 1902, from Wien. klin. Rundschau, Oct. 9, 1901.

[2] Brit. Med. Jour., Nov. 9, 1901.

[3] Lancet, Nov. 16, 1901.

[4] Lancet, Nov. 2, 1901.

[5] Brit. Med. Jour., Jan. 11, 1902, from Wien. med. Blätt., Oct. 3, 1901.

In hypertrichosis the rays should be applied several times after every hair has been shed, in order to prevent relapses. The writer has had cases under observation for more than 3 years in which no relapse has occurred. Before the hair is shed it turns white. In all other conditions where epilation is desirable the rays are the quickest and least troublesome form of treatment. No dressings are required, and no pain is experienced. Thus in tinea favosa and in sycosis the hairs act as foreign bodies, and maintain and prolong the folliculitis. In tinea favosa the whole scalp is exposed to the rays, which are discontinued immediately on the appearance of any hyperemia beyond that due to the disease. From 7 to 11 applications are usually sufficient to loosen the hairs and to produce complete baldness. Redness and all abnormal symptoms disappear in from 10 to 17 days. The rays, however, have some action beyond that due to thorough epilation, since before the hair is shed—after, for instance, 4 or 5 applications—the scalp, formerly covered with pustules and sores, becomes dry, and all new-formation of pustules ceases. The same is true of sycosis, which often yields to the rays after resisting other forms of treatment for years. The treatment is discontinued after complete baldness is attained, so that the root-sheaths are uninjured and the hair begins to grow again in a fortnight. No relapses of tinea favosa have occurred in Schiff's practice, though they have in two instances of sycosis, 2 and 4 months respectively, after the suspension of treatment. If inflammatory reaction is to be avoided, as in epilation, the strength of the current should not exceed 2 ampères, and the E.M.F. 12 volts; the tube should not be nearer to the skin than 8 or 10 inches, and the duration of the application should not exceed 10 minutes. If an inflammatory reaction is desired, as in the treatment of lupus, the strength of the current and the E.M.F. should be increased to 3½ ampères and 12¾ volts respectively, and the tube approached to within 4 inches of the skin. Parts in which the action of the rays is undesirable should be covered with a mask of lead-foil. "Hard" (that is, highly evacuated) tubes are invariably employed by Schiff, since "soft" tubes, although more active, are liable to produce undesirable and unexpected reaction.

NEOPLASMS.

Colloid Degeneration of the Skin.—C. J. White[1] records a case of this rare form of degeneration of the skin, and refers to the few hitherto (but well-known) reported cases. Besnier located the true seat of the disease in the corium itself and not in the sebaceous glands. Prior to this date the disease had been designated colloid milium. In White's case the individual lesions, seated on the backs of the hands and on other exposed parts of the body of a healthy man aged 52, consisted of flat papules from ⅛ to ¼ of an inch in diameter, rounded or polygonal in outline, exceedingly translucent and yellowish in color, soft, elastic, and almost gelatinous to the touch. An excised tumor showed the growth to consist of irregularly shaped islands of tissue bounded at intervals by capillaries

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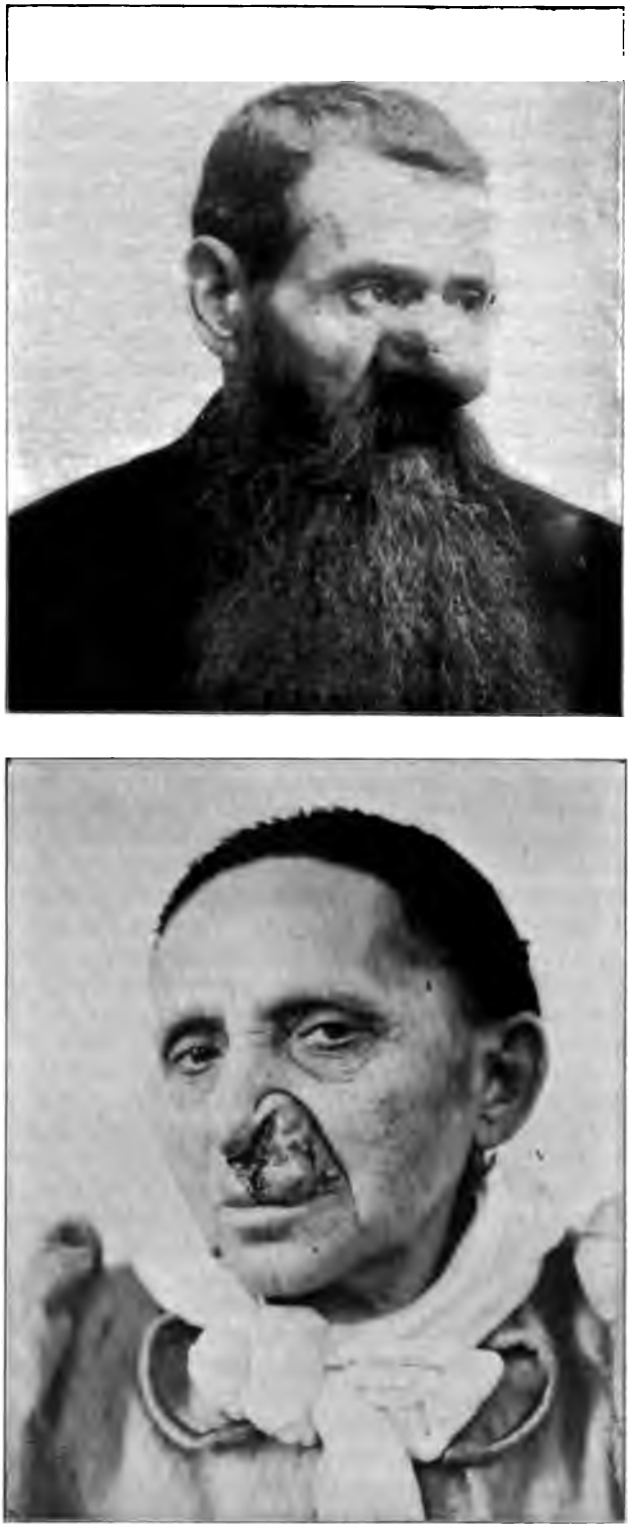
[1] Jour. of Cutan. and Gen.-Urin. Dis., Feb., 1902.

PLATE 5.



Rhinoscleroma (Allen, in Jour. Am. Med. Assoc., Dec. 14, 1901).

whose walls contained thickened masses of elacin. Within these inclosing capillary walls the true colloid material appears composed of a groundwork of fine or coarse granules. Through this homogenous mass the connective-tissue nuclei are perfectly preserved, and on approaching the encapsulating vessels many surrounding leukocytes with a rare plasma cell are found. The collacin or collastin which Unna found so abundant could not be discovered.

Xanthoma of the Eyelid.—F. J. Leviseur[1] favors the employment of electrolysis, its action being weak and under control, and the pain occasioned "very slight." The needle connected with the negative pole is inserted horizontally into the growth. The hydrates of lime, potash, and ammonia which are found on the negative pole also serve to destroy the new growth.

A Case of Fibroma Molluscum.—M. B. Hartzell[2] records the case of a young woman whose entire skin was the seat of numerous pea-, chestnut-, and walnut-sized fibrous tumors. The microscope showed the substance of the growth to consist of fibrous tissue which at the periphery was arranged in a loose meshwork, while the central portion was compact. It is probable that these growths originate either deep in the corium or in the connective tissue of the fat-lobules, and that in some cases all of the structures are involved.

Rhinoscleroma.—C. W. Allen[3] reports, with photograph, a case of this rare disease, which is seldom encountered in this country. The patient was born in Hungary and was 64 years of age. The disease began as a nodule on the upper lip and gradually by increased growth encroached upon the cavity of the nostril. It occupied the entire thickness of the lip, was circumscribed, and firm or hard. A few years later the indurated patch broke down in a gangrenous slough and was cast off, leaving denuded the bones of the upper jaw and nasal septum. At this date the general condition of the patient was such that an unfavorable prognosis as to her life was made. Later, however, the exposed bones had become wholly covered with neoplastic tissue of wavy appearance, in all probability of the same nature as the original tumor. (Plate 5.)

Granuloma Annulare.—H. Radcliffe Crocker[4] describes several cases of a peculiar form of disease with this designation. The chief characters of the disease are: papules or nodules which develop slowly and tend to form circles by aggregation or partial confluence, leaving the component parts still visible. They tend to undergo slow involution, so that the circle is broken and often only a crescent or aggregate patch is left. The inner or involuted side of the ring slopes down gradually into the normal skin, while the outer border is more abrupt, but crenate or distinctly nodular. The color may be pale red or violaceous red, and there may be a slight areola. The lesions are firm, and some of them may have a slightly warty character, while others are flat and rather suggestive of lichen planus. The distribution is chiefly on the wrists and backs of the hands and fingers, but also on the neck, and it is singular that in no less than 4 of the author's cases there have been papules on the nape near the hair-line. Under the microscope the structure appeared to be that of a granuloma—a dense mass of cells. There was very little increase in the corneous layer but the mucous layer was enormously thickened. The papillas were quite obliterated in the central portion. They were extremely indolent in their course, and were not of a common type of inflammation. The author believes that the disease belongs to a special type, which is midway between an inflammation and a neoplasm, both in its clinical and pathologic features. [Chromolithographs accompany the article.]

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[1] Med. News., Dec. 14, 1901.

[2] Am. Jour. Med. Sci., Feb., 1902.

[3] Jour. Am. Med. Assoc., Dec. 14, 1901.

[4] Brit. Jour. of Dermat., Jan., 1902.

A Case of Leprosy in an Infant Born in Minnesota.—B. Foster[1] states that in this case the father of the patient came from a leprous district in Norway 40 years ago, but that there is no leprosy in the family history. The mother was born in Minnesota. The disease was of the anesthetic type. The author believes this to be the first reported case born in Minnesota or indeed in that part of the United States, and this is the more important for the reason that it is generally supposed that all the cases of leprosy in the northwest have had their origin in some other country.

LUPUS ERYTHEMATOSUS, LUPUS VULGARIS.

Lupus Erythematosus Cured by the X-ray.—R. F. Woods[2] reports a case with a half-dollar-sized lesion on the cheek near the angle of the mouth, which had been for several years subjected to various modes of treatment. Improvement was observed after one exposure, and at the end of the sixth visit further treatment did not seem to be required. About 11 volts were used for 10 minutes, the tube being 7 inches from the skin.

Phototherapeutic Treatment of Lupus Erythematosus.—Leredde[3] reports his experience with 11 cases; all had proved rebellious to various other well-known methods of treatment. Three were cured, 2 were benefited to a great degree, 4 were being improved under the treatment and 2 showed no marked improvement.

The Etiology of Lupus Erythematosus.—Poor[4] considers the arguments urged in favor of the tuberculous origin of lupus erythematosus, but does not find that this affection occurs most frequently in those the subjects of tuberculosis or with a distinct tuberculous family history. The local and general reaction which follows tuberculin injections in lupus erythematosus only occurs irregularly, and cannot be regarded as evidence of its tuberculous nature. The presence of giant-cells cannot be regarded as proof of the tuberculous character of the disease, since these are found in other diseases in no way related to tuberculosis. The occurrence of forms transitional between lupus vulgaris and lupus erythematosus may be explained by supposing that the existence of the latter malady lowers the natural powers of resistance of the tissues and prepares the way for the invasion of the tubercle bacillus, so that there is in these cases a combination of two diseases. Lupus erythematosus, therefore, can be attributed neither to the direct invasion of the tubercle bacillus nor to the remote effects of the toxins produced by this bacillus. It probably does not have a uniform etiology, but is produced by different kinds and degrees of peripheral and central irritation acting upon the variously reacting skin of the individual.

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[1] Jour. Am. Med. Assoc., Aug. 31, 1901.

[2] Am. Jour. Med. Sci., Dec., 1901.

[3] Bull. Gén. de Thérap., Jan. 23, 1901.

[4] Dermatol. Zeitschr., April, 1901.

A Case of Tuberculosis of the Skin Following Accidental Inoculation with the Bovine Tubercle Bacillus.—Ravenel[1] records a case of tuberculosis of the skin resulting from the infection of a wound during autopsies on 2 tuberculous cows. On July 27, 1901, Dr. G., while performing autopsies on 2 cows, the subjects of experimental tuberculosis, wounded the flexor surface of one of his wrists. By September 10th there was a nodule in the skin at the seat of the wound, measuring 15 by 8 mm. This was excised and portions inoculated into two guinea-pigs, both of which developed generalized tuberculosis. Sections showed the characteristic histologic picture of tubercle and also large numbers of typical tubercle bacilli. Such cases do not settle the question of the transmissibility of bovine tuberculosis to man, but go to prove that the bovine germ finds soil and conditions in the tissues of man suitable for its multiplication. The author has reported 3 other similar cases.

Lupus Carcinoma.—Ashihara[2] records 3 cases of lupus of the face associated with carcinoma cutis. From a study of his own and 122 other cases, he states that the variety of lupus does not influence the occurrence of carcinoma. Lupus carcinoma occurs at a comparatively early age; advances rapidly; and recurrences after removal are frequent. Metastasis are comparatively rare. Operative treatment is advised in all cases.

The Present Position of the Light Treatment of Lupus.—The editor of the "British Medical Journal," January 4, 1902, goes over the ground of the treatment in general and in particular of the various forms of apparatus in use at that date, after stating that institutions for administering the light treatment have been opened on many of the larger cities of Europe, and in New York, Baltimore, and Washington. The opinion of experts may be gleaned from the discussion which took place at the meeting of the British Medical Association at Cheltenham. (See "British Medical Journal," Sept. 28, 1901, p. 849.) At the London Hospital, Sequiera records 40 complete cures out of 214 cases of lupus (with improvement in all the others), giving about 18 % of recoveries; M. Morris has had 8 cures out of 36 cases (although 6 cases were unsatisfactory and unsuccessful), giving about 22 % of recoveries; Noiré, in Paris, has had 5 cures (all in cases of localized lupus and each by 5 exposures) out of 20 cases, giving a proportion of 25 % of recoveries, while in 12 of the others there was marked improvement; and of 456 of Finsen's cases 130 are known to have remained free from recurrence for intervals varying from 1 to 5 years after cessation of the treatment. The main drawbacks to Finsen's original method are its cost and the time. Each sitting lasts 1¼ hours on, it may be, 200 or 300 different days or even more.

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[1] Univ. of Penna. Med. Bull., Feb., 1902.

[2] Arch. f. Derm. u. Syph., Bd. LVII, Heft 1 u. 2, 1901.

The objects of the Lortet-Genoud lamp and its modifications is to obtain the chemical rays near their source before dispersion, and to protect from the heat rays. Kjeldsen's lamp gives an arc light, but it differs from the ordinary arc light in having one only of its poles a rod of carbon, the other being metallic mercury. The light obtained, although feeble in luminosity, is much richer in the required chemical rays, and therefore cheaper to produce. In Bang's lamp iron electrodes replace the carbon electrodes. The electrodes may be cooled by water, either by having them hollow so that it may flow through them, or by having them suspended in a vessel containing water. Compared with other lamps the cost is small.

Phototherapy in Lupus.—Leredde[1] gives the results of his experience in the light treatment of lupus and other affections of the face. After describing the well-known apparatus used by Finsen, the writer explains the new modifications of it by Lortet and Genoud, which he has used with success. The arc lamp used is weaker than Finsen's, and the current is 10 to 20 ampères. The time of exposure by this apparatus is only 15 minutes, instead of an hour by the Finsen apparatus. Also larger areas can be treated. While admitting the good results obtained by the light treatment, he does not consider that it totally replaces the other forms of treatment, such as galvanocautery, scarification, and excision. He is not in favor of scraping owing to the frequent relapses. In lupus of the orifices he prefers phototherapy. For lupus of the mucous membranes he has devised an apparatus by which the light rays can reach the affected parts. He concludes that phototherapy is indicated (1) in lesions of the face which are rebellious to other methods; (2) in lesions of the face which would leave much scarring after total excision; (3) in certain cases of acute rosacea, rhinophyma, epithelioma of the face, and sycosis. His experience of the treatment of alopecia areata by phototherapy is not in accord with the results claimed by Finsen and others.

Lupus Treated by Blue Light.—Colquhoun[2] gives a simple method of employing therapeutically a concentrated beam of blue light, good results being obtained by daily sittings of an hour's duration. The patient sits with the back to the window, and sunlight is reflected from a mirror and focused through a biconvex lens. Placed between the subject and the lens is a bottle containing an aqueous solution of ammonio-sulfate of copper, which takes up the heat rays.

Radiotherapy of Lupus.—Leredde[3] reports that he had during the year treated 43 cases with lupus of from 1 to 35 years' duration. They had a previous record of cauterizations, scarifications, injections of tuberculin, and applications of high frequency electricity. Only one case had been cured by these remedies. Under the radiotherapy 8 were cured and 7 seemed to be cured.

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[1] Brit. Med. Jour., Nov. 16, 1901; Presse Méd., Sept. 7, 1901.
[2] New Zealand Med. Jour., Mar., 1901.
[3] Jour. Am. Med. Assoc., Feb. 22, 1902.

C. A. Greenleaf[1] describes 2 cases of this disease treated by x-rays; one was a case of direct infection from the husband to the wife, another a case of general systemic tubercular infection in which the lupus was prominent. In 3 cases all manner of treatment had been unsuccessfully used; on account of the areas involved excision seemed out of the question. Under the x-rays repair began almost immediately, recovery taking place in due time.

E. Swales[2] also employed urea together with Röntgen rays in two cases of lupus vulgaris over a period of 9 months. The dose of urea began at 20 grains and was gradually increased up to 120 grains 3 times a day. The result was that the cure was more rapid than if the rays alone had been employed.

X-rays in the Treatment of Lupus and Rodent Ulcer.—J. Startin[3] cites 5 cases, one a lupus erythematosus, and another a psoriasis, in all of which this treatment proved valuable. In 2 cases of rodent ulcer there was considerable inflammation of the skin, but the author regards this as an advantage rather than the reverse.

Lupus Vulgaris Treated by Hydrogen Dioxid.—C. H. Gunsom[4] regards this drug as valuable not only in lupus but in tuberculous abscess, considering it unirritating and harmless if not employed in full strength. He uses it in the form of a fine spray, and with most encouraging results, the granulations soon assuming a healthy appearance, the stratum lucidum becoming very active, proliferating over the granulation tissue when it reaches its level. The remedy is also valuable when sprayed on ulcers and purulent wounds. [Cases are given.]

Formalin in the Treatment of Lupus.—W. Scatchard[5] reports a case of cure with good scar of lupus of the nose of 5 years' standing from the application of equal parts of formalin (formic aldehyd, 40 %) and glycerin, preceded by orthoform powder to lessen the pain. There were during the following year at or near the original site slight recurrences, which disappeared under the same treatment. When orthoform was applied about an hour before the painting, there was no pain.

Lupus Successfully Treated with Potassium Permanganate.—Butte[6] recommends compresses saturated with a warm 2 % solution, to be kept on 15 minutes, and repeated daily. In 10 days the lesions are covered with a blackish coating and are softer and flatter. At the end of 2 or 3 months cure is usually effected. Prior to the applications the affected area should be washed with ichthyol soap or an antiseptic emulsion like the following: Hydrarg. chlor. corr., gr. v; tinct. benzoin., ♏lxxv; tinct. saponis, ℥iss; aq., ℥vij. Dupuy[7] has collected a large number of observations relative to the treatment of this disease by the same drug. Butte's method has been modified as follows: A 1 : 50 solution is applied with compresses in ulcerated cases, and in nonulcerate forms a powder is employed. The points in favor of this treatment, as set forth by Dupuy and Butte, are that it is painless, easy of application, and inexpensive. It is more rapid than scraping, and the resulting scars softer and flatter than those following most other methods. In cases in which the entire thickness of the skin is involved the method is not to be recommended. It is useful in lupus erythematosus as well as in lupus vulgaris. The staining of the tissues is of course a disadvantage. It is not applicable to the disease affecting mucous surfaces.

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[1] Buffalo Med. Jour., Oct., 1901.

[2] Lancet, Mar. 8, 1902.

[3] Lancet, July 20, 1901.

[4] Brit. Med. Jour., Feb. 22, 1902.

[5] Brit. Med. Jour., May 4, 1901, p. 1078.

[6] Jour. Am. Med. Assoc., June 14, 1902.

[7] Thèse de Paris, 1900, from Brit. Med. Jour., April 5, 1902.

Treatment of Lupus Vulgaris by Ethyl Chlorid.—C. A. Dethlefsen[1] reports a case of 12 years' duration in which there was a large ulcer with spongy granulations on the cheek extending down and overhanging the upper lip. The diseased skin was frozen with ethyl chlorid (without previous scraping) daily for a week, and later every second or third day. The freezing was succeeded by a determination of blood to the area with serous exudation and crusting. The crusts were removed before each freezing. The photographs taken a week after beginning treatment and after complete cure show a remarkable result.

A Case of Lupus Vulgaris of 12 Years' Duration Cured with Urea Employed Internally.—A. H. Buck[2] was led to use this substance in lupus, from the results obtained by Harper (see "The Lancet," March 9 and June 15, 1901) in tuberculosis of the lungs. The case was a marked and virulent one involving the face, much of which was hideously deformed by open ulceration extending deeply into the deeper structures, the nasal cavity and even the palate being perforated by the disease. There was no doubt about the diagnosis. From 20 to 60 grains of urea, together with ½ ounce doses of maltine were administered in peppermint water, t. d., beginning on March 26th, and by May 27th ulceration had nearly healed, the result being very remarkable. The first signs of improvement were noted in 2 weeks after the first dose. During the second month of treatment the improvement was so rapid and striking that "the disease seemed almost to melt away." The ulcers were dressed with ointment of iodid of lead.

CARCINOMA.

Cancer of the Skin.—J. F. Fordyce[3] considers in particular the histology of cancer growths of the skin, and concludes from his microscopic examination of numerous cases that cancers of the skin differ widely in their structure and in their power to produce general infection, and that the value of comparative methods of treatment without regard to the type of new-growth is difficult to estimate. Circumscribed, non-infiltrating cancers are readily cured by excision or caustics, and show little tendency to recur. On the other hand, chronic infiltrating rodent ulcers recur quite as often after excision as after the use of the curet, and suitable caustics. Curettage alone without the subsequent use of active caustics, like arsenic or zinc chlorid, spreads the growth by opening the lymph-vessels, and more readily permits absorption and dissemination of the cancer-cells. Caustics destroy the cell-growth which is beyond the reach of the curet, and if repeatedly used when the slightest recurrence shows itself, many infiltrating cancers may be radically cured with less destruction of tissue than by the purely surgical methods. It is in this class of cases that such brilliant results have been attained by the Röntgen rays.

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[1] Hospitals tidende, Jan. 16, 1901.

[2] Practitioner, July, 1901.

[3] Jour. of Cutan. and Gen.-Urin. Dis., April, 1902.

Treatment of Carcinoma Cutis.—P. G. Unna[1] places the Paquelin cautery in the first rank as a therapeutic agent, usually employing it superficially by singeing or stabbing the parts. It often checks the malignant process and the formation of granulation tissue. He regards resorcin as second in efficiency, used as an alcoholic solution of 5 % strength or in the form of a plaster. It seems to be more effective when combined with benzoic acid in same strength solutions. The following in plaster form is also recommended: Acidi arsenosi, ext. cannabis indicæ, aa gr. lxxv; acid salicylici, ℥v; prepared in plaster form. Under this the diseased tissue breaks down, while the healthy skin resists it for a much longer period.

Chromic Acid in Carcinoma Cutis.—D. W. Montgomery[2] speaks well of the local use of chromic acid crystals in the more superficial forms of this disease. The crystals must be very dry, and all disease tissue should be first well curetted. Sometimes it is best to wait 24 hours after scraping before applying the drug, which is placed on the wound and pressed into it, the excess in fluid form being removed. No other dressing is used, an adherent crust being formed. A lesion the size of a thumbnail will usually heal in about 5 weeks. The pain may be sharp for 15 minutes. The resulting scar is usually a good one. Twelve cases are given.

Treatment of Cutaneous Carcinoma by the X-rays.—C. W. Allen[3] believes that the x-rays will eventually be made serviceable in the treatment of cutaneous carcinoma, but at the same time he states that the method is as yet only in its infancy. He considers that the proper place of the rays is in the after-treatment or as a means of checking extension of the disease. He therefore prefers, in the first place, to follow the ordinary rules of surgical practice, and after an operation has been performed either with the knife or with caustic he strongly recommends that the rays should be applied, not, however, to the extent of producing a "burn." As a source of electric force a static machine is preferable in an institution or a private office, but in a patient's house an induction coil is more convenient, although, perhaps, inferior from a therapeutic standpoint. As to the dangers from the application of the x-rays, he believes them to have been overestimated, although it is freely admitted that in incompetent hands much damage may be done. The idea that applications of methylene-blue used concurrently with x-ray treatment interfere with the action of the latter is not credited.

Arsenic in Cutaneous Epithelioma.—Lassar[4] has treated several cases of "cancroid" with Asiatic pills. A woman, aged 62, had a nodule about the size of a lentil on the right side of the nose below the bridge. It was slightly raised above the surface and was covered with a rust-colored scab. It had appeared 3 months previously, and had slowly increased. A small piece was excised for diagnostic purposes, and proved to belong to an epithelioma in an early stage. Asiatic pills, each containing 1 mg. ($\frac{1}{64}$ gr.) of arsenious acid, were taken in first increasing and later decreasing quantities for several months, until, in all, 1 gram, or 15.4 gr., of arsenious acid had been taken. From the beginning a gradual diminution of the tumor was obvious, and finally it disappeared completely, leaving no trace except a tiny linear scar of the exploratory incision. There had been no recurrence. The arsenic produced no complications. Eight years ago the writer showed 2 cases of epithelioma and 1 of rodent ulcer before the Berlin Medical Society which were completely cured by the internal administration of arsenic. One patient, a woman, died of senile decay, aged 81, two years ago without recurrence having taken place. The patient who had the rodent ulcer is still alive and well. The third patient was a man aged 66, with a raised epitheliomatous growth on the left ala nasi. It completely disappeared under arsenic, leaving behind a small depressed scar. There had been no recurrence up to February, 1901.

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[1] Jour. Am. Med. Assoc., July 6, 1901, [2] Occidental Med. Times, July, 1901.

[3] N. Y. Med. Rec., Jan. 25, 1902.

[4] Brit. Med. Jour., Sept. 28, 1901, from Sonderabdr. aus der Berl. klin. Woch., No. 10, 1901.

Paget's Disease of the Vulva.—Dubreuilh[1] describes a case of this disease in the person of a woman aged 51. The disease began about the clitoris and gradually spread until the anterior halves of both labia majora were the seat of a large, angry looking patch, having a well-defined outline, hard, slightly swollen and raised. The surface was dark, red, excoriated, oozing, smooth, and dotted with small white spots which are raised a little above the surface and appear as islands of thickened epidermis. There was no itching, but occasional darting pains, and there was a swollen lymphatic gland in the left groin. The whole patch was excised. The lower strata of the epidermis presented the appearance of an ordinary carcinoma of the skin spread out on the surface rather than deep-seated. In some parts there were found carcinomatous nodules deeply embedded in the skin, due to the spreading of the more marked superficial disease spreading downward into the hair and mucous follicles of thé labia majora. The sweat-glands were not affected. The author believes this to be the first recorded case of the disease attacking the female genitalia.

NEUROSES, ETC.

Treatment of Pruritus.—Leo[2] states that persons suffering with pruritus often have alkaline urine, and that this is due to alkalinity of the blood. By the administration of small doses of dilute sulfuric acid he says that many cases may be cured. Jünicke[3] speaks well of treating senile pruritus by brushing the regions affected with a soft brush for 10 or 20 minutes 3 times daily. At first the itching is augmented, but diminishes as the operation nears conclusion. The sedative action of the brush is increased by the application of alcohol, used only during the first 2 or 3 days of the treatment, for after that it causes disagreeable or painful sensations.

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[1] Brit. Med. Jour., Sept. 21, 1901.

[2] Bull. Méd., Sept. 4, 1901.

[3] Bull. Gén. de Thérap., July 23, 1901.

Internal Use of Carbolic Acid in Generalized Pruritus.—J. F. Schamberg[1] advises the use of this drug according to this formula: Carbolic acid, 24 to 72 minims; glycerin, 1 to 2 drams; sherry wine, q. s. ad 3 ounces. Dose, 1 dram after meals. Several cases are referred to, improvement taking place in all cases and cure in some.

Electricity in Anal and Vulvar Pruritus.—In this form of disease Leredde[2] relates his experience with electric currents of high frequency. In 4 cases of long standing that had resisted all other known remedies he had met with success, in each case from 6 to 13 applications being required, 2 or 3 in a week, the duration of each being gradually prolonged to 15 minutes. The existence of thickening of the integument seems to be no obstacle to cure.

Pruritus Cured by Local Use of Suprarenal Extract.—F. S. Meara[3] reports 2 cases cured by the application of a strong solution of this drug to the anus and vulva. The itching in 1 case stopped for 14 hours, when a fresh application was made. The mucous membrane was dried and puckered, but this was relieved by boric acid ointment. Two months later the itching had not recurred. In the other case relief was equally prompt.

J. P. Tuttle[4] says that after bathing the anus with hot water black wash is excellent, and also the following formula: Carbolic acid, ʒij; salicylic acid, ʒiss; borax, ʒj; glycerin, ʒj. Methylene-blue, 5 %, is also well spoken of. Where there are fissures an ointment of extract of conium ʒij, ointment of stramonium ℥j, lanolin ℥j, applied at bedtime and before defecation, is well spoken of.

The following formulas for pruritus are recommended by M. B. Hartzell:[5] Carbolic acid, ʒss; glycerin, ʒij; camphor-water, ℥iv; to be mopped, not rubbed, upon the affected region every 3 hours. Menthol is regarded as next to carbolic acid in value, as in this paste: Menthol, gr. viij; powdered starch and zinc oxid, of each ʒiss; cold cream ointment, ʒv. In senile pruritus thymol as in this lotion is well spoken of: Thymol, gr. viij; liq. potassæ, ℥j; glycerin, ʒiij; water to ℥viij. In rebellious pruritus of the anus and vulva solution of silver nitrate, 10 to 40 grains to the ounce, is recommended, to be applied every second day.

The Microbacillus of Seborrhea (Sabouraud).—J. F. Schamberg[6] discusses the recent publications of Sabouraud with the view of determining the meaning of these bacilli as found in particular in the sebaceous glands of the face. He concludes that while one must admire the industry and the close powers of observation which have enabled Sabouraud to elaborate the attractive theory presented in his recent work, "Les Maladies Seborrhœiques," it appears to him that the evidence presented as to the parasitic nature of seborrhea, acne, and baldness, is by no means conclusive; and that it has not been definitely proved that the microbacillus described by Unna and Sabouraud is not a more or less normal resident of large sebaceous glands. Schamberg has repeatedly found myriads of microbacilli in the sebaceous glands of healthy skins.

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[1] Therap. Gaz., June, 1901.
[2] N. Y. Med. Jour., Nov., 1901.
[3] Merck's Archives, May, 1902.
[4] N. Y. Med. Jour., Aug. 3, 1901.
[5] Jour. Am. Med. Assoc., Mar. 8, 1902.
[6] Jour. of Cutan. and Gen.-Urin. Dis., Mar., 1902.

Tannoform as a Preventive in Hyperidrosis and Sore Feet.—Merz[1] used this drug in a large number of soldiers, concurrently with other forms of treatment. It was mixed with 2 parts of talcum and was rubbed into the skin, the results being very satisfactory. He agrees with K. Ullmann that the prophylactic treatment must extend over at least 8 days, when for weeks to come those so treated will not suffer with sweating of the feet. The best time to employ it is on going to bed. When the odor is disagreeable, this too disappears.

Iodic Purpura.—A. Stengel[2] directs attention to an uncommon manifestation of chronic iodic poisoning, characterized by marked dark blue patches of purpura of the legs, fetid breath, soreness and some sponginess of the gums, tremor of the extremities, notable moisture of the skin, rapidity of heart's action, and swelling of the thyroid gland.

LIGHT TREATMENT.

Actinotherapy in Cutaneous Medicine.—W. S. Gottheil[3] describes a machine designated the "actinolyte," manufactured in New York. It consists essentially of an arc light with the necessary arrangements of automatic action, inclosed in an iron hood. In front of the arc is a double convex condenser, adjustable for focusing by means of a rack and pinion. On rods that project in front from each side of the hood is a double convex focusing lens, also adjustable. By means of these condensers a circle of light of any size can be readily obtained. The author has used the machine with benefit in several cases, which are cited. The advantages of the method are that it is eminently conservative; it causes no radiodermatitis, ulceration, or loss of hair; it is painless; and it is effective. He concludes that we possess in concentrated light a powerful therapeutic agent.

A New Lupus Lamp.—G. G. S. Taylor,[4] of Liverpool, describes and depicts a new lamp for light treatment, which embraces all the advantages of Hedley's lamp,[5] and furthermore is self-feeding. The operator can thus control several lamps or work an x-ray apparatus at the same time. It consists of a differentially-wound arc lamp, which turns with an arc of constant length during the life of the carbons. The current is automatically kept constant at 10 ampères and is supplied from the street main (an outline figure of the lamp is given).

W. S. Hedley, of London, describes[6] and photographs a lamp which is a modification of Lortet and Genoud's now well-known lamp. Its advantages are: (1) instead of the heavy currents (80 ampères) hitherto used, only 12 ampères are required; (2) a larger area (1 to 6 cc.) can be treated at a time; (3) the photo-chemical intensity is such that an exposure of 15 to 20 minutes is sufficient to obtain the required result instead of an hour or more, as with the Finsen apparatus. (A photograph of the lamp is given.)

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[1] Practitioner, Feb., 1902.

[2] Therap. Gaz., 1902, vol. xxvi.

[3] Jour. Am. Med. Assoc., July 6, 1901.

[4] Brit. Med. Jour., Sept. 28, 1901.

[5] Brit. Med. Jour., Sept. 7, 1901.

[6] Brit. Med. Jour., Sept. 7, 1901.

Further Remarks on Finsen's Light and X-ray Treatment in Lupus and Rodent Ulcer.—M. Morris and S. E. Dore[1] discuss the disadvantages of the light treatment and the advantages of the x-rays. The disadvantages of Finsen's (light) method are the tediousness and length of time, and the small area that can be treated at a time; the elaborate and costly apparatus, and the large staff of attendants required, although the French lamp of Lortet and Genoud and others does away with most of these disadvantages. Although the authors have found the x-rays very useful as an adjunct to the light rays, they do not, except in certain cases, regard them as an adequate substitute. They conclude that Finsen's method is more reliable and apparently gives more permanent results. The effects are more easily controlled and the scar is better; the x-ray scar seeming to have more tendency to contract. This was well shown in one case where both methods were applied to different parts of the skin, the scar resulting from the x-ray being more discolored, denser, less supple, and more tense than the other, which was so fine and smooth as almost to resemble normal skin. In ulcerating cases the x-rays are effective in so far that the ulcerations heal rapidly, more so than under Finsen's light. For mucous membranes inaccessible to the light the x-rays are very useful. The danger from dermatitis and necrosis from the x-rays may be avoided by the use of very low tubes and short exposures. It is much more difficult to estimate the dosage of x-rays than it is in the case of the violet rays. Good reactions are desirable after long applications of Finsen's rays; when there is no reaction there is comparatively little benefit. The therapeutic effect of the x-rays is otherwise much greater when a reaction occurs. In ulcerating cases reaction does not occur so readily as in the unbroken skin, and may not come until healing has taken place and new epidermis formed. With daily exposures of 10 minutes, with the anticathode at a distance of about 5 inches from the patient's skin, some reaction is generally manifested after 8 or 9 applications; it often comes on 2 or 3 days after the cessation of the treatment, and sometimes with but little warning. The authors obtained their results with an electric supply of 200-volt circuit, a 12-inch coil (about a 7-inch spark gap), and a mercury jet interrupter. The advisability of employing the Finsen light method or the x-rays will depend largely on the features of the case to be treated.

Phototherapy with the Foveau-Trouvé Apparatus.—Foveau[2] describes a much simpler apparatus than that of Finsen, and which is used in the Hôpital St. Louis, Paris. It consists of a parabolic mirror with an incandescent or arc lamp in the focus, the former being joined to a "concentrating cone" which ends in two quartz plates with a chamber between them. In this chamber cold water circulates which absorbs the heat rays. For compressing the skin and rendering it exsanguine the quartz plate is pressed directly on the part of the skin to be treated. It can be installed at a cost of 200 or 300 francs, whereas Finsen's apparatus costs 4000 francs. The duration of the sittings with it is only 10 minutes against 75 minutes with Finsen's.

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[1] Brit. Med. Jour., May 31, 1902.

[2] Progrès Méd., Nov. 2, 1902.

The Therapeutic Employment of X-rays.—G. H. Lancashire[1] gives the results of his experience with numerous and diverse cases. He considers, in the first place, that the method of application is most important, and that this treatment is not to be employed indiscriminately for all cases nor in all stages of disease. He considers that the good effects of the treatment are due to the mechanical stimulation of normal cell activity, whereby morbid tissue is either thrown off or absorbed by the healthy, the process, in short, partaking of the nature of inflammation. The control of this inflammation calls for the nicest judgment. Caution is given against promoting undue inflammation with the possible result of the dreaded subsequent burn. A "soft" tube, giving plenty of green fluorescence, is preferred, and the apparatus used is like that employed by Schiff and Freund, of Vienna (with whom he studied). This consists of a resistance board so arranged as to bring the main supply (at 100 volts) down to 12 volts; a mercury break, which can be regulated so as to give a wide range of alternations. As a rule, the apparatus is worked at the rate of 600 interruptions to the minute, about 4 or 5 ampères being used, with a coil capable of giving a 12-inch spark in the air. The zone around such a tube in action can be felt by the hand to be in a high state of activity, and even at a distance of several inches a distinct "brushing" sensation can be felt. Tubes which have become too "hard" by prolonged use should be temporarily discarded. A fairly thick lead mask is much preferred to one of tin-foil, for under the latter he has seen an inflammation occur. The diseased surface should always be as clean as possible, crusts, ointments, plasters, etc., being removed. In lupus vulgaris often the first indication of improvement is the softening of the skin, causing the normal lines of the features to assert themselves. Cases of lupus suitable for x-ray applications are: (1) those in which the disease exists in prominent parts, where the mischief is too extensive for Finsen's light method; (2) some ulcerated cases; cases associated with unsightly scars; (4) some cases of disease of mucous membrane. The author's experience with rodent ulcer has been quite as gratifying as that of other observers, the ulcerated cases responding to treatment more readily than the non-ulcerous forms. The applications in these cases should be continued for some time after apparent cure.

Finsen's Method of Phototherapy in Lupus and Rodent Ulcer.—Malcolm Morris and Ernest Dore[2] obtained their best results with Finsen's method in lupus vulgaris; next in order came rodent ulcer and lupus erythematosus. In mild cases of lupus vulgaris a single application of the concentrated chemical rays, after the method of Finsen, sufficed to institute the process of repair. In cases characterized by marked crust-formation the treatment had to be reinforced by pyrogallic acid. The conditions that interfere with the treatment are marked cicatrization of the parts to be treated; pigmentation and great vascularity, preventing the rays from penetrating to the proper depth; depth of the lesions below the surface of the skin; and proximity of mucous membranes and delicate organs, such as the eyes. When the diseased area of the skin is extensive, the treatment may require a long time—a year or more. Nevertheless, the treatment is acceptable to the patient, and the satisfactory results obtained far outweigh its disadvantages.

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[1] Brit. Med. Jour., May 31, 1902.

[2] Amer. Med., Jan. 18, 1902, from Therap. Monatsh., vol. xv, No. 5, May, 1901.

The Light Treatment.—S. Bang[1] deals exclusivelv with light rays, and leaves heat rays entirely out of the article. The local effect of light rays, as applied in treatment, depends on the action of ultra-violet rays, which are most readily obtained from an electric brush light. This effect is evidenced by a hyperemia which lasts for a very long time after the influence of the light has been removed. He speaks of a so-called "latent hyperemia," that condition of the skin which follows months after the treatment has been applied, and which is recognized by a distinct redness appearing after a very slight stimulation. The actual changes consist in a dilation of the cutaneous vessels, exudation, and local leukocytosis. Later a pigment is locally deposited, probably from the red blood-corpuscles, which acts as a bar against any further effect of the ultra-violet rays. As to the general action of the light, knowledge is extremely limited. It is, however, certain that the light is of importance for our health and power of resistance. Turning to the bactericidal action of the light, he believes that we are treading on firmer ground. Apart from the strength and quality of the light, one has to take into account the absorption of the rays, and it is shown that when passed through 1 cc. of a light veal-broth the rays lose 700 times as much as they do when passing through a like column of distilled water. Comparing the electric light with sunlight, it is found that while the latter takes 1¼ hour to sterilize a certain surface growth of Staphylococcus aureus, the 30-ampère lamp, at a distance of 60 cm. (23½ inches), completes the action in 4½ minutes. The light-resisting capability of different bacteria varies considerably, and tubercle bacilli may be classified under the more resistant micro-organisms. Treatment is divided into 2 classes—a positive and a negative. The latter depends upon the assumption that certain rays exert a harmful effect, and the treatment consists in applying a light freed from these harmful rays, such, for example, as Finsen introduced for variola. In speaking of the positive light treatment, there is a considerable amount of genuine action which is due to the local and general application of light rays. One of the most important details in Finsen's method is the compression, and against the suggestion that the mechanical action of this, and not the light at all, is responsible for the beneficial effect, he quotes a case of lupus which was successfully treated by applications of only 5 minutes' duration each.

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[1] Brit. Med. Jour., Jan. 11, 1902, from Berl. klin. Woch., Dec. 9, 1901.

The Treatment of Skin Diseases by Static Electricity.—Winkler[1] states that the effect of static electricity is upon the vasomotor nerves exclusively. The various methods of using this agent are as follows: The brush, the application of which causes cutaneous anemia; Morton's currents, better termed the franklinic interrupted current, which produce hyperemia of the skin, while the spark, which is between the two as regards intensity of effect, produces cutis anserina after a short exposure, erythema, and vesiculation after a more prolonged one; local franklinization, which is to be employed when an intense effect upon the vasomotor nerves is desired, as in passive congestions, and in those affections in which there is dilation of the vessels, as in pernio, varices, acne rosacea; erythema nodosum. The interrupted current is useful in the treatment of scars, and to improve the tone of anemic and scaly skins, and in keloid. The sedative and anesthetic effects of the breeze may be used in the treatment of eczema, painful scars, fissures of the nipple, urticaria, and herpes. While a number of diseases have been treated with asserted good effect, the author utters a warning against too great enthusiasm concerning this agent. The good results obtained in eczema by various observers are especially emphasized.

The Possible Curative Effects of Radium.—Booth[2] calls attention to the possible utilization of the properties of the new element radium in the treatment of those diseases in which the x-rays have been found useful. Currie allowed the rays emitted by radium to pass through a sheet of gutta-percha and act upon the skin for 10 hours. The skin soon became red, presenting all the appearances of a burn. At the end of some days the redness had increased and ulceration occurred. Healing was not properly under way before the end of 6 weeks. Becquerel suffered from a similar "burn" accidentally produced by carrying a few decigrams of pure radium, inclosed in a glass tube, in his vest pocket for some hours. A red spot appeared upon the skin corresponding in size and shape with the tube in which the radium was contained, and soon presented the appearances of a burn. Forty-nine days elapsed before healing was complete. The author suggests the possibility that this new element may produce all the effects of the x-rays, and that its powers might be employed in medicine.

The Present Status of the Finsen Therapy.—Huber[3] gives a detailed account of the Finsen method and apparatus as used in the Finsen Institute, which he was commissioned to study by the Hungarian Minister of Education. The account of the instruments and method of using them is preceded by a short historical sketch of the origin of phototherapy and a review of Finsen's experiments with the arc-light. These experiments confirmed the results of Widmark's researches, according to which the chemical rays exert a decided influence upon the skin, the visible rays also producing some effect. It was found that the ultra-violet rays of the electric light possessed the greatest bactericidal power, and that these passed readily through rock-crystal plates, but were absorbed by glass. It was also shown that the dilation of the capillaries produced by the chemical rays lasted a long time, and could be demonstrated macroscopically a half-year after their first production. The normal redness of the uncovered skin was shown to be due primarily to the chemical rays of sunlight, and is apparently prevented by heat and favored by cold. Since 1898 powerful arc-lights have been employed instead of sunlight, since with these the source of light is practically constant and readily controlled. The chemical rays of the arc-light are concentrated and the heat rays absorbed by being passed through the so-called Finsen concentrator, which consists of a telescope-like brass tube containing four rock-crystal lenses, between two of which is a quantity of distilled water which absorbs the heat rays. In a sleeve surrounding the tube fresh water constantly circulates in order to prevent the undue heating of the distilled water. Around every lamp are four such concentrators arranged at an angle of 130 degrees, this angle having been found best in practice. By means of so-called pressure glasses, which consist of two plates of rock-crystal mounted in brass, between which water constantly circulates, the part to be treated is made as anemic as possible, since it has been shown that the blood is impermeable to the chemical rays. The treatment of the diseased parts is always begun at the periphery, so that a portion of sound skin is under the pressure-glass Since an area of not more than 2 cm. diameter can be treated at one time, it is apparent that the treatment requires a long time. It is especially important that the mucous membranes be thoroughly examined and treated; and since the concentrator frequently cannot be adapted to the mucous membranes, these are treated twice a week with the galvanocautery under cocain anesthesia. In the intervals daily pencilings with tincture of iodin or cauterization with nitrate of silver are employed. After treatment of the nasal mucous membrane sublimate tampons are inserted in the nasal cavities. Tubercles upon the lips are curetted, and when the eyelids are affected these are treated with scrupulous care. Especial care is taken that the light-rays collected by the concentrator fall vertically upon the center of the pressure-glass; and surrounding parts are covered with yellow paper or wadding wet with a watery solution of boric acid to prevent undesirable effects. The eyes are also carefully covered, for which purpose each patient has his own towel. It is important that the patient experience no burning pain, as this would indicate some derangement of the apparatus. As the therapeutic result depends upon the proper adaptation of the pressure-glasses, it is important that these be so constructed as to permit their adaptation to the places to be treated, and plane, concave, convex, round, or oval glasses are used as may be indicated. The proper degree of pressure is best produced by the hand. When the diseased areas are thickly covered with crusts, these are removed before the light treatment by means of a compound pyrogallol ointment made according to the following formula: Acid. pyrogallici, 2.0; ichthyoli, acid. salicylic., āā 5.0; vaselin, 30 0. Each treatment lasts 1½ hours, but in individuals with sensitive skins this time may be reduced one-half. Immediately after the treatment a protective dressing of a 10 % zinc ointment is applied to prevent possible infection of excoriated places. The duration of the treatment depends upon the extent and duration of the disease, the condition of the tissues at the beginning of the treatment, and the depth to which the skin is affected. The treatment is interrupted if after a considerable time no improvement is observed, or if the reaction is too severe. Besides lupus vulgaris, a number of other cutaneous affections have been treated by Finsen, such as lupus erythematosus, alopecia areata, epithelioma, acne vulgaris, tuberculosis verrucosa cutis, trichophytosis capitis, and favus, with more or less favorable results. Recurrences without doubt occur, and it is therefore necessary to subject the patient to treatment from time to time until recurrences cease to take place.

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[1] Monats. f. prakt. Dermat., vol. xxxiii, No. 10, 1901; Amer. Med., Mar. 15, 1902.

[2] Med. News, Dec. 21, 1901.

[3] Wien. med. Woch., May 17, 24, 31, June 14, 21, 28, 1902.

Cases Treated with Röntgen Rays.—Pusey[1] reports a series of 36 cases consisting of lupus, carcinoma, superficial and deep-seated, keloid, sarcoma, and pseudoleukemia treated by exposure to the Röntgen rays, in the great majority of which favorable results were obtained. While a number of failures are recorded, the results upon the whole are very encouraging, since the cases were for the most part of an unfavorable character, many of them being regarded by the surgeon as inoperable. Sections made from the cases of carcinoma showed that the x-rays produce a degeneration of the carcinomatous tissue followed by its absorption and the formation of healthy scar-tissue. The method is painless, and leaves small scars. It is available in cases in which it is desirable to save tissue and to avoid subsequent disfigurement, and in those cases in which it is impossible to remove all the diseased tissues by operation; *i. e.*, in inoperable cases. It frequently relieves pain. The author believes the x-ray treatment should be reserved for those cases in which it is inadvisable or impossible to employ the ordinary methods; he does not regard it as a substitute for operation in operable neoplasms. An exception is made, however, in the case of cutaneous cancer, in which it may be employed as a primary method of treatment. It is advised that the x-rays be used after operations for malignant disease, as a prophylactic measure, and in all operable cases of such diseases. In such cases pain may be relieved and the progress of the disease inhibited.

Atoxyl (Metaarsenous Acid Anilid) a New Arsenical Preparation in Dermatotherapy.—Walther Schild,[2] of Berlin, brings forward this drug, which contains half as much arsenic as arsenous acid. It is a white, odorless powder, of a weak saline taste, which is soluble in 20 % of warm water, of which, however, on cooling 2 % is thrown down in the form of water-clear crystals. Aqueous solutions by long standing take on a light yellowish color, and the same result follows boiling. On the other hand, atoxyl is a very solid substance, which does not show the typical arsenic reactions, but forms well-marked salts. Experience showed the author that persons do not tolerate it well by the stomach, the drug causing loss of appetite, so that it was used hypodermically, at first half a syringeful of a 1 % solution (0.005) being used, and the dose increased by employing stronger solutions. The largest dose administered was two entire syringefuls of a warm 20 % solution (= 0.4), after which there always occurred kidney disturbances, characterized by chills, usually in the evening, dizziness, headache, and tickling in the throat. Seventy-five cases are cited in which the drug was used, in all about 1500 injections, in none of which was there local irritation, infiltrations, or abscesses produced. A 20 % solution is recommended as a standard, used warm, the dose being for the first injection in quantity only 2 linear marks on the Pravaz syringe (= 0.04); with the second injection, 4 linear marks, and so on, so that by the fifth injection a full syringeful is reached (= 0.2). Further than this with the dosage it is not, as a rule, well to advance. The first five doses are administered daily; after this every other day. Cases of psoriasis, lichen, and various other diseases are cited as having been cured. The drug is preferred to the other arsenical preparations, and especially for the reason that it is well tolerated, on account of the absence of local irritation, and because it seems to be harmless. The experiments were conducted in Lassar's clinic in Berlin.

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[1] Chicago Med. Recorder, April, 1902.

[2] Berl. klin. Woch., Mar. 31, 1902.

The Salts of Cacodylic Acid in Skin Diseases.—Saalfeld,[1] of Berlin, states that the first to propose an organic compound of arsenic for therapeutic purposes was Gautier, who employed cacodylic acid and its salts. The formula of cacodyl is $As\,(CH_3)_2$. When exposed to the air, it oxidizes and forms cacodylic acid, $As\,(CH_3)_2\,O\,OH$. Cacodylic acid contains 53.3 % of arsenic, corresponding to 72 % of arsenious acid. The salt in most common use is sodium cacodylate (or arsycodile), because owing to its low degree of toxicity it furnishes a means of administering considerable quantities of arsenic without risk, being well borne in considerable doses. The author uses it in pill or in solution; each pill contains 0.025 gram (about ½ grain), and of these he gives 1 or 2 at a dose, up to 4 a day. Larger doses can, however, be given with impunity: 0.4 to 0.6 gram (6 to 9 grains) per day internally and up to 0.4 gram (6 grains) hypodermically. Another preparation is ferrocodile or cacodylate of iron. Of a 5 % solution of sodium or iron cacodylate, from 10 to 20 drops—equivalent to 0.025 to 0.05 (say ½ to 1 grain) of the drug—up to 40 drops a day are given. Hypodermic injections also are used, 1 cc. of a 5 % solution being injected at a dose. The advantages of sodium cacodylate are its efficacy, the fact that it does not disturb digestion, and that it permits the subcutaneous injection of large doses of arsenic almost without pain and without unfavorable local action. Cacodylate of iron is suitable for hypodermic injection. The disadvantages of the salts or cacodylic acid are the disagreeable garlicky odor imparted to the breath, which, however, is not constant; the extreme offensiveness of the stool; the frequent occurrence of colic and of attacks of dermatitis; and, finally, the danger of producing a toxic neuritis. It is recommended for all diseases in which arsenic is given, including psoriasis, lichen ruber planus, acne, Duhring's disease, lupus erythematosus, and the like. It does not prevent recurrence in psoriasis, but rather shows a tendency to lose its power when given in successive relapses of the disease. S. S. Cohen states that he has had excellent results from sodium cacodylate hypodermically in the various anemias, including those of malarial cachexia and pulmonary tuberculosis.

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[1] New Orleans M. and S. Jour., April, 1902, from Therap. Monats., vol. LXV, No. 6, June, 1901.

Action of Arsenic on the Skin.—H. G. Brooke[1] concludes from a series of microscopic studies that the main action of arsenic is obviously a stimulation of the growth and functions of the epithelium. The interpapillary pegs are irregularly enlarged and prolonged downward, the prickle-cell layer is increased, the granular layer always very considerably thickened, the stratum lucidum abnormally well marked, and the horny layer is altered in character and unusually pronounced. As the poisonous action of the arsenic continues, either by the further absorption or the process of elimination of the arsenic through the skin, the hyperkeratosis ceases and the horny layer becomes dry and lamellar and scales off rapidly. Usually this is preliminary to restitution of tissue, but in pronounced cases the stimulation may have been so great that the cells are unable to recover, and atrophy of a more or less complete character sets in. The papillas flatten, the cells lose their position and special characters, until the whole epithelial layer is represented by 3 or 4 rows of degenerate cells, with a last trace of a stratum lucidum and a covering of very thin, flat, paper-like lamellas. The sebaceous glands are not affected, except by general atrophy, their fatty contents apparently rendering them immune to the action of the drug.

Acetanilid-iodized Starch Powder as a Local Remedy.—T. G. Lusk[2] speaks highly of the following combination in numerous diseases, applied in the form of a powder or a paste: Acetanilid, ʒj; zinc oxid, ʒiij; iodized starch, 5%, ʒiv. It is necessary that the iodized starch be properly prepared and the acetanilid finely pulverized. Sufficient water is added to make a paint or paste, to be applied with a stiff brush. Liquid albolene, benzoin, or olive oil may be used instead of water when the application is intended for dry surfaces or ulcers; a gauze bandage may be used to prevent it from being rubbed off. When dry the powder is of a light drab color, when wet of a slate color, but when in contact with pus it turns white, showing that the iodin has been liberated. It is an antiseptic, astringent, soothing, and protective remedy, having remarkable healing properties, useful in eczema, ulcers, inflammation from all causes, including superficial burns, impetigo, sycosis, herpes zoster, and chancroids.

Hydrogen Dioxid in Pustular Diseases.—Cochart[3] praises this drug in the various diseases in which pustules occur, especially in children. He gives 15 cases of impetigo and abscess treated by the pure drug in the form of lotions. It is preferred to any other antiseptic lotion in such cases.

Phenol Used Internally in Skin Diseases.—Jay F. Schamberg[4] says that carbolic acid employed internally has fallen into disuse. In psoriasis it is an excellent remedy and can be administered in pill form for long periods, the dose varying from 5 to 10 drops, the latter very rarely causing irritation of the kidneys. It is especially useful in chronic psoriasis when the patches are not much infiltrated. If administered in solution, the following prescription is useful: Acidi carbolici, ʒiij; glycerini, f℥j; aquæ destillat., f℥v: One dram in a large wineglass of water, before meals.

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[1] Brit. Med. Jour., 1901, No. 2126, p. 860.

[2] Jour. of Cutan. and Gen.-Urin. Dis., Dec., 1901.

[3] Amer. Med., Jan. 18, 1902, from Jour. des Praticiens, April 13, 1901.

[4] Therap. Gaz., June 15, 1901.

SYPHILIS.

Syphilis and Tuberculosis.—Mauté[1] discusses the reciprocal relations existing between syphilis and tuberculosis, studying in particular the association of the tuberculides, tubercular adenopathy, and pulmonary tuberculosis with syphilis. Although cutaneous lesions do not seem to be a common port of entry for tuberculosis, a number of cases of such mixed local infection have been reported. According to Hallopeau, the influence of the one infection upon the other is inconsiderable, but in a few cases the syphilitic lesion has seemed to prepare the way for the tubercular infection which occupied the old syphilitic foci. The lymphatic glands may be the seat of a mixed syphilitic and tubercular infection; syphilis, by the profound disturbance which it produces in the lymphatic system, favoring the development of tuberculosis in the glands. Syphilis and pulmonary tuberculosis may exist together, not only in the same patient, but in the same lesion, but the frequency of such association is not yet very well determined. Syphilitic affections of the lungs present many symptoms resembling those of pulmonary tuberculosis. A cavity occurring in a patient of relatively florid aspect without any cachexia, particularly if this cavity is situated in the midst of a lung absolutely sound elsewhere, is most likely of syphilitic origin. In syphilis of the lung it is most frequently the middle portion of the right lung which is affected, and not the apices, as in tuberculosis. An argument in favor of the syphilitic nature of any lung affection is furnished by the favorable result of treatment with the remedies useful in syphilis. Although there is some diversity of opinion as to the exact degree of influence exerted, there is no doubt that syphilis favors the development and progress of tuberculosis. Tuberculosis beginning during the secondary period of syphilis usually follows a rapid course, and a pre-existing tuberculosis seems to be accelerated during this period. On the other hand, tuberculosis attacking the lungs in the tertiary period is apt to be chronic. Syphilis appears to act in two ways: as a general disease, and as a port of entry for the associated infection by the lesions which it produces in the various organs. As a general infectious disease it profoundly affects the nutrition; and the lesions of the skin, larynx, bronchi, and lungs may serve as the gateways by which the tuberculous infection may enter. In the treatment of syphilis associated with tuberculosis, it is necessary to use great care; all heroic treatment must be avoided, and mild measures must be employed. Especial care should be taken not to disturb the digestion, and the dose of mercury given should be regulated daily. Hygienic treatment and the usual remedies for tuberculosis should not be omitted.

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[1] Rev. de Tuberc., Nov., 1901.

The Bacillus of Syphilis.—Joseph and Piorkowski,[1] proceeding upon the assumption that the syphilitic virus dwells longer in the semen than in the other fluids of the body, since a symptom-free man may infect a sound woman if he impregnate her, have made a number of cultures of this fluid from syphilitic individuals, using as a culture-medium fresh, normal placenta. Upon placentas inoculated with syphilitic semen, within the first 24 hours, small, dew-like colonies, scarcely visible to the naked eye, appeared which gradually assumed a gray hue and became confluent. These dew-like colonies were found to consist of small, plump rods, club-shaped at one end and very frequently granular. The most peculiar feature was their palisade-like arrangement. In size they were from 4 to 8 μ in length and from 0.2 to 0.3 μ in thickness, and in general they had the form of diphtheria bacilli. They stained readily with the ordinary dyes, but were best stained with dilute carbol-fuchsin and gentian-violet. Attempts to inoculate bouillon and agar mediums directly with syphilitic virus failed; but colonies of the bacilli could be successfully transferred from the placenta to agar and blood-serum. In 22 cases examined from 5 weeks to 2 years after infection, the same bacillus was found in the semen. No similar organism was discoverable in normal semen, although control experiments were made in this direction. No bacilli were demonstrable in 2 patients with azoospermia; in one case with clear symptoms of syphilis and with azoospermia no bacilli were present in the semen, but could be found in the blood. From the foregoing the authors conclude that the bacilli are in some way associated only with living spermatozoa. Inoculation experiments with this bacillus upon mice, guinea-pigs, and rabbits gave negative results. In a pig inoculated with a pure culture a macular exanthem appeared about the genitalia extending into the vagina and upon the neighboring surface of the thighs; but the animal dying of swine-plague, the exact nature of this exanthem remained in doubt.

New Results of the Investigation of the Etiology of Syphilis.—Von Niessen,[2] having examined the blood from more than 100 cases of syphilis in all stages by the most varied cultural methods, concludes that the specific contagium of syphilis exists in all its phases in the blood, and may be obtained by cultures from the same. It grows best at a moderate temperature upon gelatin and bouillon, while at the temperature of the blood it is apt to undergo various changes which make its cultivation more difficult. After it is first demonstrated microscopically in the original blood-cultures as very pleomorphous nests of cocci, which in the beginning are distinguished with difficulty from the plasma derivatives of the blood, it can be luxuriantly propagated through many generations. In the beginning existing in the form of gray-white sediment, or rather pellicle, it later increases in volume and changes its color to a bright yellow. The bacillus in many of its developmental phases produces a very copious gum, and the cultures are then enveloped in a voluminous, very viscid mass. The concurrence of this gummy secretion with the gummatous syphilid is certainly not accidental. No elective method of staining the organism has yet been found. As to animal experiments, the author found swine, and, to a less degree, many kinds of monkeys susceptible of inoculation, while rodents, particularly guinea-pigs, were practically insusceptible. From previous investigations made in the Strassburg clinic the author is convinced that syphilitic exanthems may be produced in swine with syphilitic blood. In 3 swine, after an incubation of 3 to 6 weeks, a papular eruption appeared upon the flexor surface of the hind legs and upon the belly, which was so extraordinary and typical that there could be no doubt as to its specific character. In one of these animals, 6 weeks after inoculation with a fresh culture obtained from the blood of a syphilitic taken at the acme of the exanthem, a typical gumma with central softening developed. The seat of this gumma was the skin of the perineum, while the injection had been made upon the posterior surface of the ear. A further resemblance between these eruptions and those of human syphilis is the tendency to spontaneous healing and to recurrences. As the result of his experiments, the author maintains that it is possible to produce artificially syphilis in monkeys and swine by means of a contagium cultivated from the blood of syphilitics. Attempts to produce an immunizing serum were not followed by any definite results.

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[1] Berl. klin. Woch., Mar. 24, 31, 1902.

[2] Wien. med. Woch., No. 49, 1901.

Mercurial Injections and the Treatment of Syphilis.—Dieupart[1] believes that the future treatment of syphilis will be by mercurial injections because of their remarkable therapeutic activity and the slight liability to accidents. Such injections should always be intramuscular, preferably in the gluteal region. The needle should be an irido-platinum one, not steel, 4 or 5 cm. long, and should be flamed before and after each injection. Rigorous surgical antisepsis is not necessary; it is sufficient to wash the point of the injection with sublimate solution or ether. Notwithstanding their undeniable usefulness, injections of the insoluble salts of mercury present a serious inconvenience, viz., the uncertainty with which they produce mercurialization. With the soluble salts one is always master of the treatment, and need not fear emboli, stomatitis, nor the dangerous accumulation of mercury. Of the soluble injections the author finds that the bichlorureted serum of Cheron possesses many advantages. The formula for this serum is as follows: Mercuric chlorid, 0.50 cgm.; crystallized phenic acid, 2 gm.; sodium chlorid, 2 gm.; distilled water, 200 gm. The phenic acid should not be added until the solution is completely cooled, otherwise a white, insoluble precipitate forms; nor should a solution be used which has not been made at least 3 weeks, since this precipitate forms slowly even in the cold. Of this serum 20 grams should be injected into the buttock every 8 days. The advantages possessed by this serum are the following: Infrequency of the injections, freedom from danger of sudden mercurial intoxication, tolerance of the remedy, and absence of local reaction.

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[1] Jour. de Méd. de Paris, Aug. 11, 1901.

Injections of Mercurial Salts.—Leredde[1] believes that the therapeutic differences between the various salts of mercury used hypodermatically are due to the quantity of mercury which they contain. Calomel should be reserved for grave cases and for certain dermatoses; it is also a rapid means of diagnosis between syphilitic and other lesions. The author's preference in other cases is for gray oil. Rigorous asepsis is relied upon to prevent untoward accidents, the injections being made deep into the buttock. Compared with inunctions and mercury by the mouth, it avoids digestive troubles, and when well done causes neither inflammation nor embolism. As to the indications for this method of treatment, it should be employed in grave lesions which may rapidly produce irreparable injury. In benign cases its use will depend upon the patient's social condition, the condition of the digestion, etc., but it should be used as often as possible even in the mild forms of the disease. The subcutaneous method should be employed with perseverance in parasyphilitic lesions, the author having seen ocular and vesical troubles in the early stages of tabes disappear after repeated injections kept up for several months.

Gaucher[2] employs hypodermatic injections of **mercury benzoate** according to the following formula in the treatment of syphilis: Hydrargyri benzoat., 0.30; ammon. benzoat., 1.50; cocainæ benzoat., 0.075–0.15; aq. destil., 30. Of this solution 2 cc. are injected daily into the subcutaneous cellular tissue of the buttock. These injections should be continued for 2 months if necessary, suspending them only in case local troubles arise, such as the formation of nodes, or in the event of general mercurialism. This method of treatment has all the advantages of the injection of the soluble salts of mercury, and is very rarely painful if a fine needle is made use of. The indications for the use of this method of treatment are the following: phagedenic chancre, generalized cutaneous syphilids, precocious gummas, rebellious or recurring mucous plaques, precocious nervous affections, intolerance of mercury by the stomach. By the use of benzoate of mercury employed in this manner syphilitic pregnant females may go on to term and be delivered of healthy children.

Treatment of Syphilis by Hermophenyl.—Hermophenyl[3] occurs as a very fine white, amorphous powder, very soluble in water, and contains 40 % of mercury. Its solutions are unirritating and do not precipitate albumin in the cold. It has been employed as a general antiseptic and as an antisyphilitic. Reynes, who has studied the antisyphilitic properties, has treated 26 cases of syphilis with it, using the following solution hypodermatically: hermophenyl, 0.05; distilled water, 10. The injections, which were made in the buttocks and arm, produced neither abscess, induration, nor pain, the daily dose being 4 cc. In all stages of syphilis hermophenyl gave most conclusive results, the lesions being rapidly improved or cured. Given in small doses, it exercises a manifold effect upon nutrition, as shown by increase in weight and diminution of the principal elements of the urine without decrease of the total volume.

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[1] Gaz. Hebdom. de Méd. et de Chir., Feb. 23, 1902.

[2] Jour. de Méd. de Paris, Mar. 9, 1902. [3] Jour. de Méd. de Paris, Nov. 3, 1901.

The Blood in Constitutional Syphilis under the Influence of Mercurial Treatment.—Oppenheim and Löwenbach,[1] who have examined the blood of syphilitics under mercurial treatment, conclude as follows: In constitutional syphilis, before the patient has been submitted to treatment, the iron and the hemoglobin are diminished. Mercurial treatment does not exercise any regular influence upon the proportion of iron and hemoglobin. Before and during treatment the number of red corpuscles is normal and shows variations within physiologic limits. The number of leukocytes is not increased before nor during treatment. It was impossible to find iron in the serum before as well as during treatment. The results of the investigations were not influenced by the mode of administration of the mercurial.

The Blood in Syphilis, Tabes, and General Paralysis.—Sabrazès and Mathis[2] find in the primary period a slight degree of anemia and leukocytosis with an increased percentage of lymphocytes. In secondary syphilis the anemia persists and the leukocytosis is manifest in a neutrophile polynucleosis, while the number of eosinophiles remains low. Mercurial treatment increases the number of red corpuscles and the corpuscular value. In the tertiary period the blood approaches the normal. The subjects of tabes do not present any special alteration except a diminution of the corpuscular value and a very slight neutrophile polynucleosis. Those with general paralysis, in the period of euphoria, tend to an increase in the number of the corpuscles and to eosinophilia. Children with acquired or hereditary syphilis are anemic with a neutrophile and lymphocytic polynuclear leukocytosis.

The Abolition of the Pupillary Reflex to Light a Sure Sign of Syphilis.—Babinski and Charpentier[3] report 4 cases of syphilis, in all of which the Argyll-Robertson reaction was present. They believe that not only is this a certain sign of syphilis, hereditary or acquired, but that it indicates the approach of locomotor ataxia, general paralysis, or confirmed cerebrospinal syphilis, even when all other symptoms of these affections are absent.

Orthoform in Syphilis.—Bouveyron and Siraud give orthoform for the essential headache of syphilis, administering a daily dose of 2 or 3 grams, divided into 4 or 6 powders. In most cases 2 grams in the 24 hours is sufficient to relieve this obstinate headache. Usually the pain diminishes during the first night, and then disappears entirely. To prevent recurrences it is necessary to continue the drug for several days. A distinction must be made between continuous and intermittent headaches. For the intermittent variety a powder containing 0.5 gram should be taken about 1 hour before the pain usually begins, and 2 similar powders during the latter part of the night. For continuous headache 4 such powders should be taken at regular intervals. The sensation of heat or irritation which sometimes accompanies the use of the drug may be obviated by giving an equal quantity of bicarbonate of soda with each dose. Orthoform does not relieve the dysphagia of syphilitics nor the pains in the thorax, periosteum, or vital organs.

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[1] Deut. Arch. f. klin. Med., Bd. LXXI, 1901.

[2] Gaz. Hebdom. de Méd. et de Chir., Jan. 23, 1902.

[3] Bull. et Mém. de la Soc. Méd. des Hôp. de Paris, May 23, 1901; Phila. Med. Jour., Aug. 7, 1901.

Diagnostically Important Postsyphilitic and Parasyphilitic Symptoms.—Heller,[1] in a valuable and interesting paper, has brought together all the clinical symptoms by which a previous syphilis may be diagnosed after all active phenomena of the disease have disappeared. Beginning with the skin, attention is called to the scars which may result from many syphilitic lesions. It is also pointed out that the frequently repeated statement that scars upon the genitalia are pathognomonic of syphilis is an error, since any ulceration in this situation may cause scarring; and, on the other hand, the initial lesion of syphilis may disappear without producing a scar. The secondary lesions, as a rule, do not leave scars. In rare cases keloid-like formations may occur after secondary symptoms. Only tertiary syphilis produces scars upon the skin, which belong among the most characteristic postsyphilitic symptoms. These arise from the healing of ulcerating gummas, are on a level with the surrounding skin, and are smooth and slightly shining. They contain no hair nor glands, and are round or kidney-shaped, the latter form being absolutely pathognomonic. Frequently there is a more or less marked deposit of brown pigment, situated usually about the margin. Scars upon the legs associated with leg-ulcers have an especial interest. When these are multiple, round, or kidney-shaped, they should always suggest to the physician a diagnosis of syphilis. Peculiar scars are sometimes seen about the mouth in children showing a radial arrangement, which are the result of rhagades due to hereditary syphilis. These are regarded as parasyphilitic phenomena. Pigment changes in the skin frequently present important diagnostic symptoms. Leukoderma is a peculiar symptom found exclusively in the early secondary stage of syphilis, and is characterized by the appearance of pigmentless patches situated most commonly upon the throat and the nape of the neck. This symptom is about 11 times as frequent in women as in men. The nails are frequently the seat of the syphilitic process, but the diagnosis of syphilitic nail disease is often very difficult. Syphilis of the nails frequently disappears without leaving any trace; but when the matrix is affected deformity may result. The isolated papule of the nail-bed frequently causes a circumscribed defect in the nail-plate which is an almost pathognomonic postsyphilitic symptom. Nail affections in early childhood have an especial significance. Since eczematous affections of the nail are rare in this period of life, nail deformities in children may with some degree of certainty be attributed to syphilis. Examination of the lymph-glands should be omitted in no case, although their importance as affording postsyphilitic symptoms is often overestimated. Syphilitic lymphadenitis usually lasts longer than the secondary symptoms upon the skin and mucous membranes, extending into the second and third year of the disease. Of the large glandular organs of the body the testicle alone may be palpated in every part; and a careful palpation of this organ in many cases gives surprising results. Gonorrhea and tuberculosis affect usually the epididymis, while syphilitic disease of this structure is extremely rare. Syphilis may give rise to gummatous and interstitial orchitis. While the former may disappear without leaving any trace, the latter usually leaves some evidence of its existence behind in the shape of sclerosed areas.

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[1] Berl. klin. Woch., May 5, 12, 1902.

The rectum should always be examined, although this often shows no symptoms. Occasionally, especially in women, light-red spots may be observed about the anus, which are particularly noticeable because of the surrounding pigmentation, which are almost certainly the sequels of healed flat condylomas. Next to the skin and the mucous membranes, the bones are the most frequent seat of syphilis. According to the author's experience, the tibia is most frequently affected, showing circumscribed elevations, thickenings, and scars. Much less frequently the bones of the upper extremities are involved. The bones of the head are very frequently the seat of syphilitic disease which often and quickly leads to ulceration. Much less frequently the clavicle and ribs are attacked. Affections of the bones are common in hereditary syphilis. The cavity of the mouth ranks next to the skin as the seat of postsyphilitic and parasyphilitic phenomena. Some of the symptoms observed in this locality are leukoplakia, stellate scars and perforations of the hard palate, extensive scarring and consequent deformity of the soft palate. The tongue also presents postsyphilitic symptoms in the shape of scars and a peculiar atrophy producing unusual smoothness of its surface. Among the parasyphilitic symptoms observed in the mouth is the peculiar deformity of the teeth first described by Hutchinson. Distinction must be made between this deformity and that due to rachitis. Postsyphilitic symptoms occurring in the nose, apart from scars, are the results of ulceration of the nasal mucous membrane, the nasal cartilages, and bones. An unusually important symptom is perforation of the nasal septum. This results from an ulcerating gumma, and is much more frequent than is commonly supposed. Parasyphilitic symptoms are relatively infrequent in the ear. According to Politzer, a sudden deafness unaccompanied by evidence of disease in the tympanic cavity, with suspension of bone conduction of sound, is probably due to syphilitic disease of the labyrinth. Hereditary syphilis causes deafness in a proportionately large number of cases, the disturbance of the hearing occurring about the time of puberty, usually without disease of the external or middle ear. The eyes are affected in 2 % to 3 % of all cases of syphilis; and it has been shown that a large number of the diseases of the iris, the choroid, the retina, and the optic nerve occur in those who have previously had syphilis. The same is true of a large proportion of the cases of paralysis of the ocular muscles.

Acute Forms of Constitutional Syphilis.—Senator[1] discusses at some length the cases of syphilis which begin with symptoms resembling those of the acute infectious diseases. In a considerable number of cases the attack begins with fever, usually of a remittent or an intermittent type, accompanied by glandular swellings, all sorts of nervous phenomena, pains in the joints, headache, roseola, and swelling of the spleen. These cases may present considerable difficulty in their diagnosis. They may resemble more or less closely the acute exanthems, more especially measles, smallpox, and varicella. From the first-named they may usually be distinguished by the absence of conjunctivitis and the characteristic catarrh of the respiratory tract; but the diagnosis between these acute cases of syphilis and variola, varioloid, and varicella may be difficult. Typhus and typhoid fever may in the eruptive stage present considerable resemblance to acute syphilis. Two other febrile diseases which may resemble syphilis on account of the involvement of the lymphatic glands are the plague and dengue. Rheumatism may present a certain similarity to febrile cases. There are a number of affections of a more local character, which, although due without doubt to the syphilitic virus, present symptoms differing in no respect from those which accompany these maladies when due to other causes. To these belong those affections of the liver occurring in the early stage of syphilis which sometimes present the symptoms of simple catarrhal jaundice, sometimes those of acute atrophy of the liver. Another organ which may be acutely diseased from syphilis is the kidney ; acute syphilitic nephritis is rare, but undoubtedly occurs. Some other affections which, still less than those mentioned, give the impression of an acute general infection, but due to syphilis, are acute iritis, cerebral and spinal meningitis, neuritis, and strumitis.

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[1] Berl. klin. Woch., May 19, 1902.

Iodipin in Syphilis and Psoriasis.—Rille[1] has treated 2 cases of tertiary syphilis and 6 cases of psoriasis with iodipin. In the syphilitic cases daily injections of 10 cc. of a 25 % solution were made into the buttock. In the first place good results were obtained after 10 injections; in the second case 42 injections were made. In the cases of psoriasis the same solution was used with good effect.

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[1] Wien. klin. Woch., No. 30, 1901.

MATERIA MEDICA, EXPERIMENTAL THERAPEUTICS, AND PHARMACOLOGY.

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[The progress in therapeutics during the past year has been steady but without any startling discoveries. The serums and vaccinations are being estimated at their true value. The American Therapeutic Society held a meeting in May even more brilliant than its predecessor and the symposium in which the advance in the therapeutics of each specialty was presented by an able exponent was a most valuable feature of an unusual program. The increased interest manifested on all sides is an earnest of good work in the future.]

Abrin.—Salffner[1] has employed a preparation of abrin, known as jequiritol, with encouraging results in trachomatous pannus, in chronic eczematous keratitis, and in opacities due to parenchymatous keratitis, etc. An infusion of the jequirity bean was formerly employed in the treatment of trachomatous pannus, and although it gave good results it had to be discontinued on account of the severe inflammatory phenomena following an overdose. The advantage claimed for the active principle is that the dose can be accurately gaged.

Acetanilid.—Toxic symptoms have frequently followed the administration of acetanilid even in medicinal doses, but cases of fatal poisoning have been very rare. One is described by Brown.[2] A man, aged 37 years, was given 60 grains of acetanilid in 6 powders, and he took them all within a few hours. When seen by his physician, he was slightly delirious, and complained of pain in the head and in the left side of the abdomen. There were pyrexia (100.2° F.), rapid heart, marked constipation, slight jaundice, cyanosis, nausea and vomiting, and great thirst. Calomel was given in small doses followed by salts, after which copious and bloody stools were passed. The urine was strongly alkaline, and deep red—nearly black. Granular casts and albumin were present, but no biliary coloring-matter. The later symptoms were complete anuria, loss of reflexes, coldness of the extremities, and subnormal temperature. Forty-eight hours before death and 27 hours after the urine became completely suppressed, blood coloring-matter and broken-down blood-cells were constantly in the feces. Examination of the blood showed destruction of the red corpuscles, which finally were reduced to 1,166,000 per cubic millimeter, while the leukocytes were 66,450 and the nucleated red cells 22,150. The alkalinity of the blood was diminished by 80 %. The hemoglobin was 40.35 % and 32 % of normal on successive days. The red cells were unusually distorted. As the process advanced the microscopic findings approached more nearly those of pernicious anemia. The dark red color of the urine was due to hematoporphyrin, which was identified by chemical and spectroscopic examination. Death occurred on the eighth day. At the necropsy acute nephritis and intestinal catarrh were found, but nothing to account for the melena. [The number of instances of hematoporphyrinuria from ingestion of large doses or long-continued small ones is rapidly increasing.]

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[1] Arch. f. Augenheilk., XLIV, No. 4, 1901.

[2] Am. Jour. Med. Sci., Dec., 1901.

Acetozone.—Novy and Freer[1] call attention to this new antiseptic, which is said to be chemically benzoyl-acetyl-peroxid. It occurs as a crystalline body, and if heated gradually, it slowly decomposes and evaporates. It is sold mixed with an indifferent powder in equal parts, so as to facilitate handling and promote durability. As a rule, the preparation is used in saturated watery solutions, which are prepared by shaking the powder vigorously with water and filtering. Internally, it may be given in capsules containing from 4 to 5 grains each, 3 times a day. Westinghouse[2] and Abt and Lackner[3] have used acetozone as an intestinal antiseptic in typhoid with good results.

Adrenalin.—[See also Suprarenal Extract.] Houghton and Takamine[4] discuss the pharmacology of the suprarenal gland and the method of assaying its products. Adrenalin is described as a light, white, microcrystalline substance, stable, of a slightly bitter taste, and of faintly alkaline reaction. It is with difficulty soluble in cold water, and more readily in hot water. Colorless aqueous solutions are prone to undergo oxidation. The drug in becoming oxidized turns pink, then red, and finally brown. Adrenalin is readily soluble in acids, forming various salts. A fraction of one drop of the aqueous solution in the strength 1 : 50,000 blanches the normal conjunctiva within one minute. Transient paralysis is observed in frogs when adrenalin is injected into the abdominal lymph-sac, and death may result, if the dose be sufficiently large. When large doses are injected subcutaneously in dogs, guinea-pigs, etc., local irritation, sometimes followed by suppuration, is noticed. If sufficient quantities are absorbed, excitement followed by paresis of the hind limbs, accelerated respiration, and dyspnea are often noticed, and death from respiratory failure occurs. Extremely small quantities of adrenalin, when injected intravenously, produce almost instantly marked rise in blood-pressure with slowing and strengthening of the heart-beats. This rise in blood-pressure is due mainly to contraction of the muscular coat of the arterioles from the local action of the substance on their walls, to stimulation of the heart-muscle itself and stimulation of the vasomotor center. The slowing of the pulse is due to stimulation of the vagus center. Except for the very fleeting influence of adrenalin on the circulation, its action might be compared to that of digitalis. Abel[5] contends that the commercial preparation known as adrenalin is not a chemical individual, but probably a mixture of native and reduced epinephrin with traces of foreign substances rich in nitrogen. Langley[1] finds that suprarenal extract causes a brief but active **secretion of the salivary and lacrimal glands.** This is not affected by degeneration of the postganglionic fibers of the superior cervical ganglion or by small doses of atropin, but the secretion is stopped by large doses of atropin. The extract has no certain effect on the secretion of sweat, but increases the secretion of bile. It acts differently on different involuntary muscular organs, causing, for instance, inhibition of the bladder, stomach, and gall-bladder, and contraction of the anal sphincters, uterus, and blood-vessels. The theory that the extract acts on sympathetic nerve-endings breaks down in various instances.

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[1] Pharm. Centralbhalle, July 31, 1902.
[2] Buffalo Med. Jour., Aug., 1902.
[3] Therap. Gaz., Oct., 1902.
[4] Jour. Am. Med. Assoc., Jan. 18, 1902.
[5] Bull. Johns Hopkins Hosp., Nov., 1901.

Blum,[2] Croftan,[3] and Herter and Richards[4] have shown that the injection into the abdominal cavity of dogs of adrenalin solution, 1 : 1000, produces **a well-marked glycosuria.** Croftan attributes this glycosuria to the presence of a ferment in the suprarenal gland, but Herter and Richards show that boiling the solution of adrenalin, which would be expected to destroy any diastatic ferment, does not prevent the glycosuria after experimental injection. Furthermore, they found in 2 dogs that died after the injections granular degeneration in the cells of the islands of Langerhans of the pancreas. This last discovery, in connection with the finding by Opie, Herzog, and others of profound changes in the islands of Langerhans in diabetes, suggests that some cases at least of diabetes may be dependent upon lesions of the suprarenal bodies.

Marple[5] considers that adrenalin has only an occasional temporary **value in ophthalmology.** It has no curative effect and may aggravate the condition if used too often. There is no inflammatory ocular condition in which he thinks it of any value, and its advantage in case of hemorrhage is not great. He thinks he has seen one or two cases in which iritis may have been caused by it. He suggests that such a bloodless condition of the eye as it produces may favor infection by weakening resistance. He therefore employs it very little in most operations. For slight operations on the lids he uses it because after he is through it enables him to dismiss his patients more promptly.

Harrison[6] has employed adrenalin internally with considerable advantage **in cases of uterine myomas.**

Keyes[7] concludes from his experience in the past 2 or 3 years that adrenalin has only a limited value **in genitourinary diseases.** He employs it sometimes in meatotomy to avoid hemorrhage, but the effect on the bleeding is only temporary. He has found it to fail utterly in any considerable hemorrhage from an acutely congested or granulating prostate, and in cancerous cases of vesical hemorrhage it has not been satisfactory.

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[1] Lancet, Mar. 8, 1902.
[2] Deut. Arch. f. klin. Med., Oct. 30, 1901.
[3] Amer. Med., Jan. 18, 1902.
[4] Med. News, Feb. 1, 1902.
[5] N. Y. State Jour. of Med., July, 1902.
[6] N. Y. State Jour. of Med., July, 1902.
[7] N. Y. State Jour. of Med., July, 1902.

Elsberg[1] is convinced that the addition of adrenalin chlorid in proportion of from 1 : 20,000 to 1 : 5000 of the solution for local anesthesia has a distinct value **in minor operative surgery,** in that it does away almost entirely with the oozing of blood from the wound. As adrenalin is a cardiac stimulant, it has the additional advantage that it will counteract the depressing effect of cocain or eucain. Moreover, by keeping the local blood-vessels firmly contracted for a number of hours it prevents the congestion, and hence the pain, which is so likely to follow after the anesthetic effects have worn off. [Inasmuch as the vasoconstrictor effects of adrenalin antedate and are much more marked than the cardiac stimulant, great caution should be employed in its use when there is cardiac weakness either permanent or temporary.]

Agurin.—This preparation is chemically a double salt of theobromin sodium and sodium acetate. It is claimed to be superior to the well-known diuretin (theobromin sodiosalicylate) in that it contains more of the active base (60 %) and is free from the unpleasant effects caused by sodium salicylate. Holle,[2] who has made a careful study of the drug, concludes that agurin deserves to be considered a good diuretic; that it is well tolerated, its administration in powders being preferable to that in tablets; that the daily dose is from 15 to 45 grains; that combination with digitalis increases its diuretic action, that its effect frequently persists for several days; that it gives the best results in cardiac disease and in dropsical effusions with sound kidneys; that it has but little influence in advanced nephritis; and that it causes no increase in the heart's action.

Alcohol.—Goldberg,[3] from a series of experiments undertaken to determine the **influence of alcohol upon the natural resistance to infection,** draws the following conclusions: Doves, which are naturally immune against anthrax, become subject to infection after moderate doses (2 cm. to 3 cm.) of 40 % brandy, which produce only transient alcoholic intoxication but do not result in the death of the animal. Chronic alcoholic intoxication diminishes the natural resistance of doves to anthrax. Small doses of alcohol repeatedly given to doves infected with fatal doses of a culture of anthrax do not save the lives of the birds, and only exceptionally prolong their lives in comparison with control doves; sometimes they cause apparently earlier death of the birds. These observations of Goldberg are in part confirmatory of those of Abbott,[4] made upon rabbits.

Kelynack[5] presents a paper on the **relation of alcoholism to tuberculosis.** The experiments of various investigators are noted, showing that alcoholized animals are more readily infected by many organisms and that alcohol produces a marked negative chemotaxis; that it is difficult to render such animals immune, and that through influence on nervous structures the tissues, and especially the lungs, are prone to invasion by tubercle. Of fatal cases of alcoholic neuritis, 80 % showed pulmonary tuberculosis. In 121 cases of common cirrhosis of the liver 23 % had tuberculosis.

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[1] Amer. Med., Mar. 1, 1902.
[2] Therap. Monthly, June, 1902.
[3] Centralbl. f. Bakt. u. Parasitenk., I. Abth., vol. xxx, 1901.
[4] Trans. Assoc. Amer. Phys., vol. xi, 1895.
[5] Med. Press and Circ., Aug. 7, 1901.

Meltzer[1] states that all experiments with alcohol upon animals serve as a warning against giving the drug in too large doses to human beings. He believes that alcohol, in not too large doses, does not affect blood-pressure, but effects **changes in the distribution of the blood,** which may prove of great importance to a patient with an infectious disease, especially pneumonia or typhoid fever. Alcohol also protects the patient against excitation by stimulating the normal inhibitory mechanism of the body. He believes that while alcohol in health is often a curse, in disease it is mostly a blessing.

Ott[2] has studied a patient with tuberculosis associated with distinct fever, and reaches the conclusion that alcohol in fever, as well as in health, tends to decrease the destruction of albuminous tissue to a degree equal to that of isodynamic amounts of carbohydrate. There is an extremely low excretion of nitrogen on the first alcohol day. This, Roseman has also observed, and thinks that it is due to the effect of the alcohol upon the kidneys. Ott agrees with this view, more particularly because of the coincident reduction in the amount of urine. Further, alcohol definitely interferes with the absorption of the food-nitrogen. Practical conclusions cannot be drawn from these results at once, but the author thinks that one may consider that **alcohol is not a satisfactory foodstuff,** because we have better at our command; but that in moderate amounts in febrile subjects it has no unfavorable effects, and particularly in chronic diseases of the lung, especially when associated with depression, it may have a very satisfactory influence.

Cushny[3] maintains that the **chief therapeutic value of alcohol is in its depressant action.** This action has a definite effect in allaying the subjective symptoms, relieving the nervous strain, and promoting the rest and comfort of the patient; its action in this way is not surpassed by any other drug. The action of alcohol on the respiratory system is a matter of dispute, but there is no question as to its effect on the circulation. He calls attention to two points bearing on the usefulness of the drug as a food in diseased conditions. One is that alcohol must be used in large quantities in order to be of any value as a food, for half a pint of whisky is equal to about 2 ounces of fat. This amount of whisky, however, is sufficient to produce marked cerebral symptoms, and, therefore, its action as a food may be dismissed under ordinary conditions. In certain diseased conditions the amount of alcohol is better borne, and heroic measures are sometimes required. It may, therefore, be of some value under such conditions. In conclusion, he sums up his remarks by saying that **alcohol** deserves a place in therapeutics **as a narcotic,** to a less extent as a stomachic, and in certain conditions as a food.

Concerning the **nutritive value of alcohol,** Benedict[4] states that alcohol is rapidly and completely absorbed in the stomach. In moderate doses it is entirely oxidized in the body, but a small proportion being excreted unchanged. But it does not build muscular or adipose tissue, thus failing in one of the most important food functions. It does, however, furnish a supply of heat by its oxidation. Excessive doses of alcohol lower the body-temperature. The most accurate experiments give no positive proof that alcohol in moderate doses influences proteid metabolism any more than does fat. It frequently prevents the body-proteids and fat from consumption. In many cases, in moderate doses, it has a retarding action on many vital processes. The multitude of other materials of undisputed nutritive value renders the use of alcohol as a food unnecessary.

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[1] Boston M. and S. Jour., July 17, 1902.

[2] Arch. f. exper. Path. u. Pharm., Bd. XLVII, Hefte 3 u. 4.

[3] Boston M. and S. Jour., July 10, 1902.

[4] Boston M. and S. Jour., July 10, 1902.

Lusk[1] reviews the history of **alcohol as a beverage,** quoting largely from the literature. He concludes that it flavors food, acts as a narcotic, producing exhilaration followed by depression. It reduces the power to do physical and mental work. It causes an increased flow of all the digestive juices. In large doses, however, it acts as an irritant, causing dyspepsia.

From a study of the local effects of carbolic acid followed by applications of alcohol, Wallace[2] concludes that **alcohol checks the action of carbolic acid** because it is a good solvent of the latter. In order to remove completely the local effects of carbolic acid, the application must follow quickly, *i. e.*, within 5 minutes; since the longer the interval, the less evident is the antidotal action. Ether and chloroform are also antidotal, but are less suitable than alcohol because their irritant action conceals their antidotal effects, and, moreover, owing to their volatility they fail to penetrate sufficiently into the skin to remove the phenol present. By experiments on animals Wallace was able to show that alcohol possesses no antidotal action after phenol has been absorbed. McDonald[3] reports a case of carbolic-acid poisoning in a young girl, in which he used alcohol successfully as an antidote. The amount of carbolic acid swallowed was over an ounce, and when seen, 25 minutes after the occurrence, the patient was in collapse and was comatose. Recovery promptly followed upon washing out the stomach with half a pint of alcohol and administering strychnin and nitroglycerin hypodermatically.

Antiepileptic Serum.—Ceni[4] presents a study of the therapeutic possibilities of the blood-serum of epileptics. Eight cases of severe idiopathic epilepsy were treated with their own serum and with that of other patients. Of these, improvement is said to have occurred in all, and in 2 cases there seemed to be a practical cure. Ceni's essay was awarded the Craig Colony prize.

Antiplague Serum.—Pinto[5] reports the results of the preventive antiplague vaccination undertaken at Campos, Brazil, during the recent widespread epidemic there. Of 1803 persons who were vaccinated, only one acquired the disease, and he had it very lightly. In one district a person was encountered who refused to be vaccinated, and he was the only one attacked. Many of the vaccinated persons lived or were in contact with plague patients, and nearly all were of the poorer classes, the very ones most predisposed to the disease.

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[1] Boston M. and S. Jour., July 10, 1902.
[2] N. Y. Univ. Bull., April, 1902.
[3] Fort Wayne Med. Jour., Feb., 1902.
[4] Med. News, Mar. 8, 1902.
[5] Therap. Monthly, Mar., 1902.

Vassal[1] reports that during the epidemic of bubonic plague on the Ile de la Reunion 18 patients were treated with antiplague serum, with a mortality of 38.8 %. The mortality was 80.3 % among 51 patients who were not treated with the serum. He attributes his success to the **large doses employed,** the average being 169 cc. for each patient. He injected as much as 40 cc. directly into the veins, in some cases without the least disturbance.

Novy[2] reports a case of bubonic plague which developed in a young man working in the laboratory of the University of Michigan. The symptoms were of the pneumonic type and the onset was extremely rapid and violent. Yersin's antipest serum was given with immediate good effects. Within 24 hours the patient received 120 cc. of serum, 4 cc. being injected intravenously. Recovery was complete.

Antipneumococcus Serum.—Sears[3] presents a study of 12 cases of pneumonia treated with serum combined with other measures. The mortality was 25 %. No ill effects were observed other than those sometimes seen after the use of diphtheria antitoxin. The extravagant claims as to the effect of the serum on special symptoms receive but little support from the author's experience. Goldsborough[4] reports 9 cases of pneumonia treated with the serum. Two of the patients died. He believes that the serum has a decided curative effect. In 447 cases of pneumonia treated with antitoxic serums, which the author has collected from the literature, the mortality was 16.5 %.

Antiscarlatinal Serum.—Leyden[5] reports his observations on the treatment of 14 cases of scarlet fever with the serum from convalescents. The results were quite favorable. In 5 cases improvement was marked, while in 9 it was slight. In general, the duration of the disease was shortened, and in none of the cases did nephritis follow. He used 10 to 20 cc. of the serum, but intends in his future experiments to increase the dose to 40 cc.

Antistreptococcus Serum.—Luedeking[6] credits the failures of antistreptococcus serum to polymorphism and variability of streptococci; the fact that few septic processes in man are pure streptococcus infections; the tardy employment of the remedy; and the inertness of many of the serums that are on the market. He reports several cases of the use of this serum with advantage, and holds that its early administration in fitting cases of streptococcus infection will prove life-saving. In the author's experience the injection of the serum has no special dangers. Lenhartz[7] reports a number of cases of septic endocarditis in which he used Marmorek's serum. He believes that the patients were made worse instead of better by it. Cooper and Ogle[1] report a case of infectious endocarditis treated with antistreptococcus serum. While the temperature was favorably affected by the injections, they were not successful in averting a fatal issue.

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[1] Bull. de l'Acad. de Méd., May 20, 1902.
[2] Am. Jour. Med. Sci., Oct., 1901.
[3] Boston M. and S. Jour., Dec. 12, 1901.
[4] Jour. Am. Med. Assoc., June 28, 1902.
[5] Münch. med. Woch., Jan. 18, 1902.
[6] St. Louis Courier of Med., April, 1902.
[7] Münch. med. Woch., July 16, 1901.

Antitoxin of Diphtheria.—Siegert[2] reviews the statistics of the 5 Children's Hospitals in Vienna from 1886 to 1900, where 17,626 children with diphtheria were treated. In 1894, when antitoxin was first used, the death-rate fell markedly, even among those with intubation and tracheotomy. While over 2000 died from 1892 to 1894 out of 4894 patients, from 1895 to 1897 only 817 died out of 4143 patients.

Netter[3] reiterates that preventive injections of serum have **a pronounced prophylactic effect** and have never occasioned any serious accidents. The immunity does not commence for 24 hours and wears off by the end of 3 or 4 weeks. He believes that in schools, hospitals, etc., when a case of diphtheria occurs, the other children should receive preventive injections, and, even in the absence of diphtheria, the injections should be given in wards in which measles and scarlet fever are treated, since they ward off diphtheric infection, which is always serious as a complication of these diseases. Richardiére[4] also makes a rule of giving patients in the measles ward of the Hôpital des Enfants Malades prophylactic injections of antidiphtheric serum. In 1899 there were 15 cases of diphtheria with 9 deaths in the ward; in 1900, 15 cases with 12 deaths; and in the first 5 months of 1901 there were 5 cases with 2 deaths. The author then began to give every child admitted to the ward a preventive injection (5 to 10 cc.) of antidiphtheric serum, and since this treatment was instituted no case of diphtheria has developed.

Geffrier and Rozet[5] believe that injections of antitoxin may cause trouble, even in small amounts; that as good, if not better, results follow the injection of small doses, and that fewer accidents follow small than large doses. Large doses, they believe, aggravate renal symptoms. Thirteen cases with histories of **anuria following the injection of serum** are given in full, 4 of them personal observations. When a child arrives, even before the result of cultures is known, from 5 to 20 cc. are injected as routine treatment, according to the age of the infant. The throat is washed with a bichlorid solution daily, as long as the membrane exists. Out of 410 patients thus treated, 45 died. Albuminuria was noted in 25 % of the cases and paralysis followed in 10 %. Geffrier and Rozet do not advocate the injection of serum, even in small doses, as a prophylactic measure. Jump[6] discusses the **duration of immunity** produced by immunizing doses of diphtheria antitoxin and reports 5 cases, from which, with the experience of others, he feels warranted in deducing the following conclusions: That as diphtheria antitoxin is practically harmless, all exposed persons should receive an immunizing dose in proportion to age. That 250 units should be given to children under 2 years and 500 to all others. That the immunity will last for at least 3 weeks, provided a reliable antitoxin is used. That all exposed persons should be removed from infected surroundings, either by thorough disinfection of their own quarters or by removal to other places. If this be impossible, the immunizing dose should be repeated every third week.

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[1] Brit. Med. Jour., May 10, 1902.

[2] Jahrb. f. Kinderheilk., Jan., 1902.

[3] Bull. de l'Acad. de Méd., Mar. 18, 1902.

[4] Gaz. Hebdom. de Méd. et de Chir., Feb. 27, 1902.

[5] Arch. de Méd. des Enf., Feb., 1902.

[6] Phila. Med. Jour., Jan. 11, 1902.

Stanley[1] has made a study of the **eruptions caused by diphtheria antitoxin** in 500 cases of diphtheria treated at the Northwestern Hospital, London. Of the patients, 112 had eruptions. Of these, 58 were erythematous in character; in 15 there was combined erythema with urticaria; in 30 the eruption was an urticaria, 6 were scarlatiniform, and 3 morbilliform; in 17 there was a transient early erythema and an urticaria, usually at the seat of the injection. The period of onset was usually during the second week after the giving of the antitoxin. The typical eruption is a marginate erythema accompanied by some malaise and a rise in temperature of about 3° F. The rash usually lasts from 2 to 5 days, occasionally a few hours only. Desquamation may occur. The appearance of the rash did not influence the course of the disease either favorably or unfavorably. Le Gendre[2] reports the case of a child, 3 months old, who presented a generalized polymorphous eruption, with edema of the face and prepuce, a quarter of an hour after the injection of antidiphtheric serum. This edema persisted for 12 hours and then disappeared. Guinon[3] reports a similar case.

Konarzsheoski[4] employed **diphtheria antitoxin** in a number of cases of **whooping-cough,** all other remedies having failed to give relief. He found that 2 or 3 injections of 1000 units each were sufficient to limit the disease 2 weeks and to prevent the paroxysms. No untoward effects were observed.

Talamon[5] reports 50 more cases of **croupous pneumonia treated by diphtheria antitoxin,** with a mortality of 14 %, just as in his first series of 50 cases. The average age of the patients was 47½ years, 30 being between 40 and 78 years of age. Thirty-six were chronic alcoholics, 13 of these being inveterate drinkers. Five of the alcoholic cases died, 3 with delirium tremens. The other two deaths occurred from meningitis, one in a phthisical patient, the other in a man who drank moderately. Out of the 43 patients who recovered there was but one complication (one metapneumonic pleurisy). The case-histories of patients of 77 and 78 years of age who recovered are given in detail. Talamon concludes that the treatment of lobar pneumonia by diphtheria antitoxin, while indicated in all cases, gives especially good results in persons over 50 years of age.

Antitoxin of Tetanus.—Ager[6] reports a fatal case of acute tetanus (period of incubation 5 days) treated with antitoxin. Möllers[7] reports 4 cases of tetanus treated with antitoxin, the remedy having been given in each case within 30 hours after the recognition of the first symptoms, and in large doses. The cases were severe ones, but were free from fever. All the patients died. The author believes that while the value of the treatment cannot be stated very definitely as yet, antitoxin should be used, whenever possible, as a prophylactic measure in the case of wounds that are likely to be followed by the disease. Pfeiffer[1] reports 22 cases of tetanus and has collected 93, which he reviews. He states that the mortality of the cases treated by Behring's antitoxin was 52.7 %, while it was only 36.3 % in 88 cases treated by Tizzoni's serum. It seems evident, he remarks, that tetanus in Italy runs a milder course than elsewhere. Omitting the 21 cases reported by Italian physicians, the mortality is 46.2 %. In 14 of his personal cases in which no specific treatment was instituted, the mortality was 50 %. Leyden[2] states that the subarachnoid injection of antitoxin has been tried 11 times in man. Of the 11 patients, 6 were not saved, but the last 5—although very severe cases—all recovered, including a new case described by Leyden in this communication. The technic of the operation is the same as for lumbar puncture. The object of the spinal injections is to reach the toxin localized in the ganglion-cells of the anterior horns. It is further stated that Pfaundler has recently reported 3 cases of tetanus neonatorum treated in this way, in which the beneficial influence of the injections was unmistakable.

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[1] Brit. Med. Jour., No. 2146, 1902.
[2] Gaz. Hebdom. de Méd. et de Chir., Mar. 27, 1902.
[3] Ibid. [4] Russki Med. Viestnik, vol. IV, No. 4, 1902.
[5] La Méd. Moderne, Mar. 26 and April 2, 1902.
[6] Med. Rec., Oct. 12, 1901. [7] Deut. med. Woch., Nov. 21, 1901.

Antityphoid Serum.—As a prophylactic measure against typhoid fever, vaccination with a sterilized culture of typhoid bacilli has given very encouraging results. Birt[3] reports the results obtained with typhoid vaccine during an epidemic of typhoid fever in Harrismith, South Africa, from September, 1900, to September, 1901. During this period 1210 persons suffered from the disease. Of these, 947 were not vaccinated and among them the mortality was 14.25 %. On the other hand, 263 who had been inoculated with typhoid vaccine 6 to 18 months previously contracted the disease and gave a mortality of 6.8 %. The disease among the inoculated was considerably modified in severity, the cases among the unvaccinated having been of a very severe type. Crombie,[4] from an examination of 112 officers who had been inoculated against typhoid fever and invalided from South Africa, concludes that vaccination offers a smaller liability to attack of the disease in the long run, but that there appears to be a greater liability to infection on exposure soon after the operation of vaccination.

Petruschky[5] claims to have obtained good results in the treatment of typhoid fever from injections of a preparation of dead typhoid bacilli, which he calls **typhoin.** In every instance, according to the author, the severity of the disease was mitigated by the treatment.

Apocynum Cannabinum.—Goloubinine[6] has found this drug very efficacious in cardiac disease. He states that it decreases the pulse-rate, diminishes arrhythmia, raises the blood-pressure, increases the secretion of urine, removes albuminuria, and lessens the frequency of the respiration. He gives 5 drops of the aqueous extract 3 or 4 times a day, combining it with an equal quantity of cannabis indica to prevent gastric disturbances. [If this method of preventing digestive disturbances shall prove to be useful, the value of the remedy will be greatly enhanced by the removal of the greatest obstacle to its employment.]

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[1] Zeit. f. Heilkunde, XXIII, 2, 1902.

[2] Therap. d. Gegenwart, Aug., 1901.

[3] Brit. Med. Jour., Jan. 11, 1902.

[4] Lancet, May 3, 1902.

[5] Deut. med. Woch., Mar. 20, 1902.

[6] Jour. des Praticiens, Aug. 17, 1901.

Apomorphin Hydrochlorate.—Coleman and Polk,[1] from a study of the action of apomorphin in 300 cases of alcoholism, reach the following conclusions: To obtain a hypnotic action the drug should be given hypodermatically. It is best to begin with a small dose—$\frac{1}{40}$ grain—and to repeat this or give a slightly larger dose within a short time. Further doses should not be given after vomiting occurs, until several hours have passed. Doses repeated in 2 or 3 hours have but little beneficial effect. The administration of the drug should not be repeated in patients who are weak. The duration of the hypnotic action is only a few hours, and when the patient awakes his condition is practically unchanged, except in "ordinary drunks." The best results are obtained from apomorphin when it is followed in 2 or 3 hours by some recognized hypnotic, as bromid, chloral, paraldehyd, etc. Solutions of apomorphin are unstable and should always be freshly made for use. The best results were obtained in "ordinary drunks" and in cases verging on delirium tremens. But in some of these cases the drug had no effect whatever. The administration of apomorphin to patients in delirium tremens is, in the author's experience, without beneficial results, and may even be attended with danger from its depressant action.

Argyrol.—Barnes and Hille[2] call attention to a new silver salt which they have prepared by combining silver with a proteid derived from wheat. They have given it the name of silver vitellin or argyrol. The high percentage of silver in the compound, its extreme solubility, and its intense penetrating action on the tissues constitute its chief scientific points of interest. It contains 30 % of silver (more than three times the amount in protargol). Its solubility is quite remarkable, 1 ounce of it being completely soluble in a dessertspoonful of water. Solutions of argyrol do not coagulate albumin nor precipitate chlorids; it therefore cannot be chemically changed by contact with mucous membranes or their secretions. As an indication of the penetrating power of the compound, Martin found that by immersing a thick strand of catgut in a solution of the salt for a few hours the gut became impregnated through and through with the silver. Christian,[3] from a study of the **action of argyrol in** 48 cases of **gonorrhea,** concludes: that it is absolutely free from any irritating properties, solutions as high as 5 % causing no discomfort; that the gonococci on and beneath the urethral mucous membrane are rapidly destroyed; that the amount of urethral discharge is in a majority of cases at once lessened in a marked degree; that the actual duration of the disease is shorter than is obtained by the use of any other salt of silver. In the author's case 38 were cured in from 2 to 4 weeks. [This remedy seems to offer a decided advance in silver therapy.]

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[1] Amer. Med., Mar. 8, 1902.

[2] Med. Rec., May 22, 1902.

[3] Med. Rec., vol. LXII, 1902.

Arrhenal.—This preparation is closely allied to sodium cacodylate, from which it differs, however, in being a monomethyl sodium arsenate, while the regular sodium cacodylate is a dimethyl sodium arsenate. Gautier[1] has used arrhenal with most favorable results in tuberculosis, especially in apyretic cases. In nearly every instance, it is said, the patient gained in weight and strength. The maximum good was obtained when daily doses of ¾ grain were given. As the drug has neither odor nor taste, it is readily taken.

Aspidium.—Gotthilf[2] reports a case of poisoning from the ingestion of 2½ drams of extract of aspidium without subsequent catharsis. The symptoms were headache, anorexia, vertigo, and apathy, followed in 48 hours by coma, a rapid thready pulse, dilation of the pupils, increased reflexes, and tonic spasms of the entire body, especially of the arms. Recovery followed under stimulant treatment. The author dwells upon the importance of removing the drug with castor oil, etc., to prevent absorption and subsequent poisoning, as in this case. [The literature seems to show that almost any cathartic but castor oil is preferable.]

Aspirin.—This is a compound produced by the action of acetic anhydrid on salicylic acid. It is said to resist decomposition in the stomach and to be broken up in the intestines into nascent salicylic acid and acetic acid, the latter subsequently combining with alkalies to form sodium and potassium acetates. In a recent discussion before the Berlin Clinical Society, Ewald and Senator spoke very favorably of aspirin. Ewald has employed it extensively in various nervous disorders, especially in migraine, influenzal attacks, and angina, with great satisfaction. He regards the drug as preferable to other salicylates because it is followed by less depression and sweating. His experience has led him to think that the course of rheumatism is shortened by the administration of aspirin and that recurrences are also less frequently induced. Merkel[3] refers to the beneficial influence of the drug in gouty cases, in which he recommends that it should be given in the afternoon, at hourly intervals, in doses of 15 grains, 4 to 5 times, but not in alkaline mineral waters, which may produce a certain amount of decomposition. It is stated in a report by Wielsch[4] that Kropil and Gazert have observed depressing effects in several cases of typhoid fever shortly after its administration, and for this reason it should be given cautiously in this disease as well as in the fever of phthisical patients. Singer[5] believes that there is no evidence that aspirin actually reduces fermentation in the intestine. He bases his belief upon the fact that the indigo of the urine is often increased under the use of the drug. [Tinnitus, cardiac weakness, and renal manifestations do not seem to be less frequent than with corresponding amounts of salicylic acid. It is, however, better tolerated by the stomach.]

Atropin.—A number of reports have appeared during the last year bearing on the use of **atropin in large doses** in cases of intestinal obstruction. Aronheim reports a case of acute ileus, with great pain in the left hypogastrium, in which $\frac{1}{20}$ grain of atropin was followed in an hour by relief from the pain, and later by the passage of a large, semisolid mass of feces. Pritchard[1] reports a case with similar symptoms in a man aged 62 years. One thirty-third of a grain of atropin was given in 2 doses, 4 hours apart. This was followed by the passage of much fecal matter and a large gall-stone.

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[1] Bull. de l'Acad. de Méd., Feb. 25, 1902.
[2] Münch. med. Woch., vol. XLVIII, p. 1096, 1901.
[3] Münch. med. Woch., No. 9, 1902.
[4] Wien. med. Presse, No. 5, 1902.
[5] Zeitschr. f. klin. Med., Bd. XLIV, Hefte 1 u. 2, 1901.

Feinberg[2] reports a case of a woman, 67 years old, who was suffering from complete intestinal obstruction for 5 days, without apparent cause (there were no indications of mechanical obstruction) Three injections of atropin (0.002 gm.) during the day, followed by rectal injections, brought no relief. On the following day, however, 5 profuse evacuations occurred and the woman recovered. The author considers **atropin indicated in cases of obstruction due to atony,** the beneficial effect of the drug being probably due to its action on the center of defecation. He concludes, however, that, in view of the comparative harmlessness of the treatment, it should be employed in all obscure cases. Failing to obtain results with atropin, surgical intervention should be invoked. In a case somewhat similar to that reported by Feinberg, Medem[3] also used atropin with success.

Weber[4] concludes, from a study of the cases in the literature and his own experience, that previous treatment with opium is the explanation of the **inefficacy of small doses of atropin** in the treatment of ileus. Thirty-nine cases have been reported treated with atropin, and only 4 failed to respond to the injections. He believes that atropin treatment is not contraindicated in any form of ileus. Even in cases of incarceration which require surgical intervention, the atropin relieves pain, and after operation induces a copious harmless stool. Ileus appearing after an abdominal operation should be treated with atropin and not with opium.

Gebele[5] raises a warning cry against **the too free use of this remedy,** since it too often obscures the clinical picture, and, far from being infallible, it prevents the surgeon from selecting the proper moment for operation. It is, according to this author, only in the paralytic or spastic form of obstruction that internal treatment has any effect, and here small doses of morphin have the same effect as atropin, and are to be preferred. In mechanical obstruction atropin is still less indicated. Ripperger[6] states that atropin injections appear to influence a certain number of cases of ileus very favorably, but the method requires further testing. It may be tried in cases of dynamic ileus; cases of strangulated ileus or incarcerated hernia should be excluded from this treatment; operation is here required, but if the patient positively refuses operation, or the conditions are absolutely unfavorable, experiments may be tried with atropin. If exact diagnosis is impossible, and strangulation cannot be absolutely excluded, exploratory laparotomy is to be preferred before atropin treatment. Diatchenks[1] reports 2 cases of volvulus and 1 of intestinal paralysis in which atropin was used without effect.

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[1] Ibid.

[2] Vratch, Dec. 22, 1901.

[3] Vratch, Dec. 22, 1901.

[4] Deut. med. Woch., Jan. 6, 1902.

[5] Münch. med. Woch., No. 33, 1901.

[6] N. Y. med. Monats., May, 1902.

Boric Acid.—Rinehart[2] records 2 cases of poisoning by boric acid. The first was that of a man, aged 38 years, who was given 5-grain doses of boric acid by the mouth every 4 hours for posterior urethritis. Two days later there followed extreme weakness, and an erythematous rash beset with papules and vesicles developed on the back of the hands and between the fingers. The pulse was weak but not accelerated. The symptoms subsided slowly on withdrawing the drug and reappeared on resuming it. The second case was that of a man aged 50 years upon whom suprapubic lithotomy had been performed. The bladder had been washed out daily with a saturated solution of boric acid, and 5 grains of the drug had been given internally every 4 hours. Ten days after the operation there appeared about the wound and over the hypogastrium an eczematous rash. Albuminuria and weakness were prominent symptoms while the rash lasted, and at the height of the eruption there were nausea and fever. Rinehart concludes that in view of the above facts the use of boric acid as a food preservative should be discontinued.

Bismuth-borophenate.—Auerbach[3] reports a number of cases illustrating the value of bismuth-borophenate as a surgical dressing. He concludes that for the dressing of wounds which are, or have been rendered, aseptic it is second to no other preparation of its kind. In small and not very deep wounds it may be employed as a dusting-powder or as bismuth-borophenate collodion. When crusts are not desirable, as in burns and scalds, the drug may be applied as a paste (25 % to 50 % in glycerin) on lint.

Bromipin.—This is a 10 % combination of bromin and oil of sesame. Spratling,[4] superintendent of the Craig Colony for Epileptics, speaks favorably of its use in epilepsy. It is a heavy oil, and in some cases it may be found difficult of digestion. According to the author, it is best given in the form of an emulsion, like the following: bromipin, 4 ounces; syrup, 4 ounces; spirit of peppermint, 4 drams; mucilage of acacia, enough to make 16 ounces. Of this the dose is from 1 to 3 tablespoonfuls 3 times a day, an hour or so after meals. Bromipin is said not to cause bromic acne, and, in the form of an emulsion, to be nutritious, feeble asthenic patients gaining in weight under its use. One disadvantage of its use at the present time is its cost, which is about 5 times that of the bromids. Wassing[5] has also found bromipin of value in epilepsy, especially in persistent forms of the disease. [Owing to the complaints of patients as to its physical properties, its long-continued use is beset with difficulties.]

Bromocoll.—This is a combination of bromin, tannin, and gelatin, containing about 21 % of bromin. It is claimed that the combination passes into the intestines unaffected, but in the presence of alkaline secretions is broken down, liberating the bromin in some nonirritating form. Reich and Ehrke, who have tried the drug in a number of cases of epilepsy, conclude that it approximately equals potassium bromid in strength; that it is pleasant to take even in large doses; that gastric and intestinal disturbances do not occur; that acne is seen only exceptionally after large quantities, and then is slight; that nervous symptoms are also rare; and that bromocoll is indicated when other bromids are not well borne.

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[1] Bolnitchnaia Gazeta Botkina, Feb. 27, 1902.

[2] Therap. Gaz., Oct. 15, 1901.

[3] Therap. Monthly, April, 1902.

[4] Jour. Am. Med. Assoc., May 3, 1902.

[5] Medico, No. 12, 1902.

Caffein.—Cushny and Van Naten[1] have made a careful study of the action of caffein on the mammalian heart. They find that the effect of caffein on the dog's heart appears to consist in (1) acceleration of rhythm without further change; (2) a shortening of the movements, commencing in the auricle and spreading to the ventricle, and, in large doses, (3) auriculoventricular arrhythmia terminating in fibrillary contractions of the auricle and finally of the ventricle. The first phenomenon appears to be due to stimulation of the most irritable part of the heart, the so-called excitomotor apparatus, and as no further change in the movements is seen, the action of the drug at this stage appears to be confined to this area. The second phenomenon may be due in part to the acceleration, and may thus be considered a secondary effect of the increased irritability of the excitomotor area; but it may also be ascribed to the action of the drug on the muscle of the auricle and ventricle, and may thus indicate that the influence of the drug has extended to these less susceptible parts of the heart. The third stage of arrhythmia is due to the ventricular irritability having been so greatly increased as to give rise to an idioventricular rhythm. When this has attained a sufficient height, it leads to fibrillary contraction of the ventricles; the previous appearance of these in the auricle appears to indicate that the stimulant influence spreads to this before it reaches the ventricle. The action of caffein on the heart thus appears to consist in a descending stimulation which begins in the excitomotor area at the junction of the auricle and great veins, and extends into the auricles and finally to the ventricles. The experiments do not support the idea that the nervous apparatus of the heart is involved in these effects. Digitalis, like caffein, affects only the heart-muscle, but in the case of digitalis the earliest changes seen are in the **strength of systole and extent of diastole** in the ventricle and auricle. These parts are thus affected sooner than the excitomotor area, whereas the stimulation exercised by caffein begins in the excitomotor area and descends to the auricle and then to the ventricle, and its effects on the rhythm (as far as they are caused by direct action on the heart) are of secondary importance. Further, the primary changes induced by digitalis are not so much evidenced by **increased irritability** of the parts as by **increased contractility and lessened dilation** (increased tone), while there is no evidence of such a change in the late stages of caffein-poisoning, in which the ventricle is directly affected.

Calcium Chlorid.—Silvestri[2] proclaimed in 1898 the great value of calcium salts in all kinds of hemorrhagic affections, especially scorbutus. He administered the drug by the mouth and rectum. He has since applied this method of hemostasis in urgent cases by the intravenous route, and reports 4 in detail. He injected 100 cc. or 150 cc. of a 10 % solution of calcium chlorid, and repeated the dose in 12 hours when necessary. One patient was a girl of 13 with hemorrhage from the intestines on the fourteenth day of typhoid fever. The hemorrhage ceased in 35 minutes after the injection. Another patient was a woman of 42 with severe menorrhagia, which subsided in less than an hour after the injection. The same result followed in a case of hemoptysis and in one of hemophilia. The injection failed to arrest the hematemesis in a case of gastric ulcer, which proved rebellious to injections of gelatin later.

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[1] Arch. Internat. de Pharm. et de Thérap., vol. IX, 1901.

[2] Gaz. degli Osped., April 10, 1902.

Calcium Monosulfid.—Abogado[1] confirms the efficacy of the treatment of diphtheria with calcium monosulfid recommended by Tissot, of Switzerland. According to this author, it cures mild cases without further medication, and in severe cases proves a most valuable adjuvant to the antitoxin treatment. He established the efficacy of the drug in 110 cases of associated diphtheria, in 21 cases of mild, pure diphtheria, and in 13 severe, pure cases. None of the last two groups died. He usually administers ⅙ of a grain every hour up to 1 year of age, every half hour between the ages of 1 and 3, and every 15 minutes between the ages of 3 and 5, suspending the medication whenever the breath exhales the characteristic odor very intensely.

Camphor.—Hoffmann[2] has found camphor very efficacious in overcoming the "abstinence" symptoms resulting from the withdrawal of morphin in those who have become addicted to its use. He gives ⅔ grain of camphor by the mouth from 2 to 5 times a day according to the patient's condition.

Camphor Bromid.—Bourneville[3] has been using camphor bromid for several years in the treatment of chorea, insomnia, hysteria, delirium tremens, etc., and has found it exceedingly efficacious, especially in epilepsy with vertigo. He published in 1895 5 cases of the latter treated successfully with it, and now describes in detail 3 more. Two were cases of symptomatic epilepsy and one was a case of idiopathic epilepsy. All were clinically cured, with no recurrence of the seizures or vertigo to date, several years in one case and two in another after the institution of treatment. [This practically confirms many observations as to the value of the monobromated camphor of the U. S. Pharmacopœia.]

Carbolic Acid.—Enriquez and Baur[4] report a case of **acute tetanus** in which the convulsions resisted treatment with chloral and antitetanic serum. Under treatment by subcutaneous injections of 200 grams of simple serum containing 3 cc. of a 2 % solution of carbolic acid, the seizures, after 8 days, disappeared, but the patient died finally of pneumonia. The authors believe that large injections of weak carbolic acid serum are preferable to simple injections of carbolic acid. Enriquez[5] reports another case of severe tetanus in a woman, in which recovery followed subcutaneous injections of weak carbolic acid serum. Musgrave[1] reports a case of tetanus in which the patient received 1.2 grains of carbolic acid in a 2 % solution every 4 hours, or 7.2 grains daily, hypodermatically. The treatment was continued for 2 weeks without untoward effects. The patient recovered. Kellog[2] reports a case of tetanus in a young girl which developed 7 days after she had received a lacerated wound of the hand. The treatment consisted chiefly in the administration of 30 minims of a 1 % solution of carbolic acid hypodermatically, every 3 hours. Recovery followed in about 3 weeks. Henry[3] describes a case of very severe tetanus in a lad, aged 19, in which a total of 303 grains of carbolic acid was administered within a week. No untoward effects followed this large amount of carbolic acid, but, save for some lessening in the frequency and severity of the convulsions, the treatment was without effect and the patient died the tenth day of illness.

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[1] Cronica Med. Mexicana, Dec. 1, 1901.

[2] Therap. Monats., July, 1902.

[3] Arch. de Neurol., July, 1902.

[4] Gaz. Hebdom. de Méd. et de Chir., Dec. 26, 1901.

[5] La Semaine Méd., Dec. 25, 1901.

Josias[4] concludes from two series of experiments on goats that the **Bacelli method** (carbolic acid injections) has a markedly unfavorable influence upon experimental tetanus in goats. [Bacelli's method of treating tetanus has been before the medical world for several years and its value seems pretty thoroughly established.]

Mutschler[5] reports 2 cases of **facial anthrax** successfully treated with injections of carbolic acid around the focus of infection and with wet bichlorid dressings.

Chaulmoogra Oil.—This is an unofficial preparation expressed from the seeds of the *Gynocardia odorata*, a tree growing in the East Indies. Both the crude oil and its active principle, gynocardic acid, are used in medicine. The dose of the oil usually advised is 5 to 10 minims in capsules, and of the active principle, ½ to 3 grains. Hollopeau[6] comes to the following conclusions: (1) **Lepers** treated with large doses of chaulmoogra oil, either by intramuscular injections or by the mouth, may show so much improvement as to be considered cured. (2) More frequently the disease continues to show itself, but usually in a benign manner. (3) In some cases marked local and general exacerbations occur in spite of the treatment. (4) The effect of chaulmoogra oil, although beneficial, cannot be compared with that of mercury and iodin in syphilis or of quinin in malaria. (5) The hypodermic method is to be preferred if it is well borne; it is, however, a painful method. (6) The comparative effects of the various products supplied under the name of chaulmoogra oil should be studied. Elliot[7] reports a case of leprosy of 2 years' duration in which considerable improvement followed within 18 days under chaulmoogra oil. Twenty drops of the oil were given thrice daily, in milk. Unna[8] believes that chaulmoogra oil is the best drug for the general treatment of leprosy. [The use of this oil as a local application for painful muscles, resulting or not from traumatism, seems to be less known than its value warrants.]

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[1] Merck's Archives, Jan., 1902.

[2] Med. News, Mar. 1, 1902.

[3] Am. Jour. Med. Sci., Dec., 1901.

[4] Méd. Moderne, Oct. 16, 1901.

[5] Ann. of. Surg., Oct., 1901.

[6] Epit. Brit. Med. Jour., Nov. 16, 1901.

[7] Jour. Cutan. and Gen.-Urin. Dis., No. 233, 1902

[8] Therap. der Gegenwart, July, 1902.

Chloretone.—Cappelletti[1] gives the result of his trial of chloretone as a hypnotic in 25 cases of mental disease. The duration of sleep varied from 2 to 5 hours, according to the dose and the condition of the patient; sleep came on in 37 minutes to 2½ hours, and was tranquil. From 15 to 23 grains is sufficient, as a rule, but in very restless subjects 30 grains may be given. The pulse and respiration did not appear to be notably affected by the drug and the reawakening was not accompanied by unpleasant symptoms. The digestive system as a rule was unaffected, and the same could be said of the secretions.

Chinosol.—This is the potassium salt of a compound of oxychinolin and sulfuric acid. Nottebaum[2] considers it an especially valuable preparation because it combines antiseptic with styptic properties. Clinically, he has observed the latter property in the case of a wound of the head with profuse parenchymatous hemorrhage and in 2 cases of severe epistaxis. The chief drawback to its general use is the readiness with which it is precipitated from solution. In disinfecting the hands with chinosol, distilled water must be used after washing with soap, as the latter precipitates the disinfectant.

Chlorinated Lime.—Zeuner's[3] experience with 100 cases of **ulcer of the leg** treated with a 1 % solution of chlorinated lime has convinced him that this simple, old-fashioned remedy is the most effective of all at our command. He applies it on several thicknesses of gauze and then wraps the leg in a tricot bandage, commencing at the toes. The purulent secretions, it is stated, dry up at once, the lesions present a clean granulating surface, and the formation of skin is exceptionally perfect.

Tichy[4] treats **extensive burns** of the second and third degree by applying oil for the first day, then pricking the blisters and applying compresses wet with a solution of 2.5 to 5 parts of chlorinated lime with 5 parts of spirit of camphor, in 990 parts of water. The compresses are kept constantly wet with this solution and are not removed for from 4 to 6 days. Tichy has never witnessed any inconveniences from this treatment in the 40 cases in which he has employed it; on the contrary, healing occurred in all with remarkable rapidity.

Chloroform.—Huchard[5] maintains that **accidents from chloroform anesthesia** are no more frequent in persons with lesions of the heart or aorta than in cases of other affections. A cardiac or aortic affection is, therefore, no contraindication to chloroform under the following conditions: that it is not in the acute stage of an infectious process; that the organ affected is not too much degenerated; that it is not in the asystolic or dyspneic stage nor exhibits symptoms of adhesion of the pericardium. In case of cardiac or aortic disease, the chloroform should be administered in small, progressive, and continuous doses until the lid reflex is diminished. Chloroform well made and well administered does not kill, said Sédillot, and the maxim still holds good.

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[1] Rif. Med., Dec. 3, 1901.
[2] Deut. med. Woch., Aug. 15, 1901.
[3] Deut. med. Woch., May 15, 1902.
[4] Deut. med. Woch., July 17, 1902.
[5] Bull. de l'Acad de Méd., Feb. 11, 1902.

In the discussion[1] which followed Huchard's communication the speakers agreed with the author of the paper that there is **no more danger from chloroform in compensated mitral disease** than with the heart intact. Bucquoy ascribed death under chloroform to reflex action on the centers of respiration and circulation, and stated that in the majority of cases death had occurred in subjects with sound hearts. Lucas-Championnière administers the anesthetic on this principle, starting with a few whiffs of ethyl bromid to deaden the reflexes. Le Dentu remarked that Huchard's conclusions represent the average opinion of most surgeons nearly everywhere now, as it has been tentatively established of late. He urged, however, closer study of the deaths that occur a few hours or days after the anesthesia, as he believed they were due to the heart in many instances. He quoted Finney's experience in 142 narcoses on subjects with heart disease. This author found that the anesthetic was well borne in cases of valvular disease, while myocarditis required special surveillance. Bucquoy and others called attention to the greater tendency to syncope and sudden death in cases of lesions of the aortic orifice.

Guyon[2] remarked that he had frequently had occasion to operate on subjects between 60 and 90 years of age, and although **atheromatous affections** are not rare at these ages, the **chloroform was perfectly tolerated.** He has lost 2 patients under operation, and in both the heart was the seat of fatty degeneration, hence he warns against this condition, but affirms, on the other hand, that chloroform can be administered to patients even when they are known to have exhibited symptoms of atheroma or angina pectoris. He thinks it possible to avert syncope, even in extreme anemia, by the Trendelenburg position.

Brouardel[3] stated that he had served as a medical expert in 25 cases of sudden death from chloroform. One was a child with an enlarged thymus and one a woman who succumbed to pulmonary embolism after reduction of a fracture of the femur. None had any evidence of valvular disease, but in 3 the heart was loaded with fat, although the cardiac muscle was not altered. In one patient the heart was in actual fatty degeneration. The subjects were between 13 and 52 years of age, 9 men all under 47. In 14 cases the operation had not commenced; in 3 it had just commenced. Death was attributed by the surgeons, in all these cases, except one, to **cardiopulmonary syncope.** Renal lesions were frequent in the various subjects. From the medicolegal standpoint, Brouardel believes that the fatalities can be compared only to the sudden deaths which occur from blows in the region of the larynx, sometimes very slight injuries. The danger is not exclusively in the administration of the anesthetic agent; it lies also in the innate or acquired susceptibility of the patient.

Pannas[4] attributes sudden death under chloroform to a **bulbolaryngeal contraction** of the vocal cords, from contact of the former with the glottis. Although the mechanical phenomena of respiration continue, the heart soon ceases to beat, and the death is erroneously attributed to cardiac syncope. He states that the anesthetist should devote his chief energies to surveillance of the free intake of air. He should listen closely to hear the laryngotracheal respiratory souffle. He believes that if this precaution is observed death will never occur. He has performed 15,000 operations without a mishap. The persons for whom chloroform is inclined to be dangerous are not those with heart disease, but nervous persons with peculiarly intense reflexes easily induced and long persisting.

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[1] Bull. de l'acad. de Méd., Feb. 18, 1902.

[2] Bull. de l'acad. de Méd., Feb. 25, 1902.

[3] Ibid.

[4] Bull. de l'acad. de Méd., Mar. 4, 1902.

Barette[1] states that but 3 patients died out of 6000 chloroform anesthesias. One, a man of 23, with a previous history of alcoholism, died during operation, his **heart suddenly ceasing to beat in diastole.** Nothing could resuscitate him. Autopsy showed a very fatty heart.

Embley,[2] as the result of a series of experiments on dogs, concludes that the heart-muscle itself is very sensitive to the poisonous effects of chloroform. **Chloroform raises the excitability of the vagus,** especially in the early part of its administration. This increased excitability of the vagus mechanism is due to the action of the chloroform on the centers. The inhibitory action is more intense and fatal, because it is exercised upon a heart the spontaneous excitability of which is already diminished. Chloroform vapor not stronger than 1.5 % in the air administered to morphinized dogs after a period of mild excitation, slowly depresses the vagus excitability; above 2 % in the air it may occasion dangerous, persistent inhibition. Section of the vagi or atropinization absolutely abolishes sudden heart arrest from chloroform, the heart under such conditions failing slowly with rapid pulse. Under these conditions the respiration fails when the blood-pressure falls sufficiently, and invariably long before the heart stops. The central medullary vasomotor system is stimulated, at any rate for a time, by chloroform. The effects of chloroform on the peripheral vasomotor mechanism are paralytic. Failure of respiration is due to fall in blood-pressure, and with a good blood-pressure respiratory failure is practically impossible. Chloroform in the early stages of the administration should be given slowly and in a strength of less than 1 % until the initial excitability of the vagus mechanism gives place to diminished excitability.

Bonreau[3] reports **1200 cases of chloroform narcosis without a death.** He uses a folded handkerchief, the chloroform being administered drop by drop, plenty of air being given first. The tongue is drawn forward. Narcosis was usually complete in from 8 to 10 minutes. Renal and cardiac diseases were not regarded as contraindications. The Trendelenburg position, the author believes, is of advantage in chloroform anesthesia. In 12 cases syncope occurred, and it was necessary to resort to artificial respiration.

Crouch and Corner[4] believe from their statistics, which cover more than 3000 anesthesias in the St. Thomas Hospital, that **ether** in its way **is less safe than chloroform.** There were 10 cases of respiratory complications, all from ether administration, the patients all having been in good condition and the anesthetic being given under favorable conditions. All were trunk cases, and such as required bandaging that of necessity restricted the chest and abdominal movements. In one case of bronchopneumonia the outcome was fatal. In 600 patients who had chloroform none developed any respiratory trouble. The authors, therefore, recommend chloroform in all long operations on the trunk.

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[1] Jour. des Praticiens, Mar. 1, 1902.

[2] Brit. Med. Jour., April 19, 1902.

Rev. de Chir., No. 5, 1901.

[4] Lancet, May 24, 1902.

Zematsky[1] advocates the use of **ethyl bromid prior to the administration of chloroform** as an anesthetic. A few drops of ethyl bromid are sufficient to produce sleep, when the chloroform may be substituted. According to Zematsky, in less than a minute the patient passes successfully through the phases of analgesia and anesthesia to loss of consciousness, with entire absence of excitement. The author observed no accidents of any kind in an experience with 3000 subjects.

Cinchonin Sulfate.—Talley[2] calls attention to the power of this drug to check nasal and nasopharyngeal secretion. He has found from 4 to 6 grains, 3 or 4 times a day, sufficient to control the excessive secretion in coryza, hay-fever, and allied conditions.

Cinnamic Acid.—Heusser,[3] of Davos Platz, has previously published 22 cases of tuberculosis treated with cinnamic acid, and now adds 60 more to the list. The results have convinced him that this method of treating tuberculosis, within the limits established by Landerer, is entirely harmless and is more effective in inducing cicatrization and healing in cases of uncomplicated tuberculosis than any other remedy yet recommended for the purpose.

Mann[4] reports 7 additional cases of tuberculosis treated by intravenous injections of **sodium cinnamate.** He states that he has been using the drug for the past 6 years with very satisfactory results.

Cocain.—Reichert[5] presents an elaborate paper on the **effects of cocain on temperature.** He concludes from his research that the rise of temperature caused by cocain is due to an increase of heat-production, and that the latter depends upon two actions—one, upon the cortex, causing motor excitement, and the other, upon the caudate thermogenic center, by which heat is produced independently of motor activity; that cocain possesses very little power as a thermogenic in animals lightly curarized, because of both motor quietude and the depression of some other portions of the thermogenic apparatus; that cocain is absolutely without thermogenic power in animals in which the pathways of thermogenic and corticospinal motor fibers have been cut, as after sections of the spinal cord at its junction with the bulb and of crura cerebri; that cocain is effective as a thermogenic when only a small portion of the caudate center is left intact with the parts below; that cocain and morphin are direct antagonists in their action upon the caudate and cortical centers which are directly or indirectly involved in the changes of temperature and heat production.

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[1] Vratch., XII, 34, 1901.
[2] Therap. Monthly, Feb., 1902.
[3] Correspondenzbl. f. Schweiz. Aerzte, Jan. 1, 1902.
[4] Phila. Med. Jour., Mar. 1, 1902.
[5] Phila. Med. Jour., Aug. 2, 1902.

Copper Acetophosphate.—Liegeois[1] follows out the teaching of Mendini in employing copper salts in chlorosis, amenorrhea, and cervical lymphadenitis. The acteophosphate is the form preferred, and in many of the cases under observation for the past 25 years marked improvement in the blood-condition has resulted.

Creasote Carbonate.—Van Zandt[2] states that he has been using this drug in the treatment of **croupous pneumonia** since 1890 and has lost but one case; in this the drug was absolutely ineffective. While he has seen one or two other cases in which the drug has failed, he believes that only a small percentage are unaffected, and that a large number are shortened or aborted, while almost all the rest are mitigated by it. He has been giving it in doses of 7½ to 10 grains every 3 hours. He rarely used any other medication except strychnin occasionally, and an anodyne in the beginning of painful cases. Thomson[3] reports 18 cases of pneumonia treated with creasote carbonate. Of these, only one died. The cases were mostly average ones, but 2 or 3 were decidedly severe. He is of the opinion that the drug may exert a special effect upon the disease, as shown by the temperature curve after its administration. He recommends that it be given in doses of 15 grains every 2 hours, day and night. He has observed no injurious effects from its use in these doses. Wilcox[4] reports 33 cases of pneumonia treated with creasote carbonate without a death. The disease terminated by lysis in 9; by crisis in 24. Crisis occurred on the sixth day in 1, seventh in 2, eighth in 9, ninth in 6, tenth in 3, eleventh in 2, and on the twelfth in 1 patient. In 2 patients above the age of 70, lysis occurred. Of 3 alcoholic subjects, in 2 lysis and 1 crisis was noted. Two cases of double pneumonia terminated by lysis. Weber[5] has also used creasote carbonate successfully in 9 cases of pneumonia. Meitner[6] reports 75 cases of non-tuberculous diseases of the respiratory organs in children, including **laryngitis, bronchitis, and bronchopneumonia,** in which he used creasote carbonate with advantage. He recommends 15 grains daily for children up to 6 months of age and 23 grains daily up to 1 year of age. [The results reported by so many observers are so distinctly favorable that pessimism as to medicinal treatment in these conditions should speedily become unfashionable.]

Demulcents.—To determine whether the group of drugs known as **demulcents really possess the property of allaying irritation,** Tappeiner[7] conducted a large number of experiments on animals and human beings. A distinctly diminished irritability of motor nerves by chemical salts was found in the presence of mucilaginous substances. The same can also be said of sensory nerves, since pain appeared later and was less intense than in control experiments. The mucous membranes of the eyes and intestines gave less evidence of inflammation when demulcents were added. On the other hand, the cathartic action of drugs is enhanced, since their irritating effects are prolonged, owing to slow absorption. It was also found that this absorption is less active in the stomach and the upper part of the intestines, and that this holds true for water alone. All these experiments tend to prove that the time-honored custom of using demulcents to allay inflammation and irritation is based upon scientific facts.

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[1] Jour. des Praticiens, vol. vi, 1901.

[2] Medicine, Feb., 1902.

[3] Med. Rec., Feb. 1, 1902.

[4] Am. Jour. Med. Sci., Sept., 1902.

[5] Med. Rec., Nov. 2, 1901.

[6] Allg. med. Cent. Zeit., Jan. 22, 1902.

[7] Arch. internat. de Pharm. et de Thérap., vol. x, Nos. 1 and 2, 1902.

Digitalis.—Jacobi[1] states that he has long urged the use of digitalis in small doses for various **cardiac conditions and tuberculosis,** giving from 4 to 6 grains daily or its equivalent, for weeks or months, or even years, without any hesitation. He finds no cumulative action in the use of the drug in these doses, nor do patients become accustomed to its effect so as to reduce its efficiency. He also mentions as of value small doses of digitalis in chlorosis, in which disease he combines the drug with iron and vegetable purgatives.

As opposed to Jacobi's teaching, Porter[2] contends that the **prolonged use of digitalis acts damagingly on the heart-muscle,** both by its action as a muscle-poison and by its interference with cardiac nutrition through the circulation. Digitalis, he thinks, is of service temporarily in certain cases by improving the mechanical action of the heart, as in mitral insufficiency and stenosis, but in aortic lesions there seems to be no good reason for using it at any stage. It is contraindicated, according to Porter, in fatty degeneration or in enfeebled condition of the heart-muscle. It should only be used to influence the heart and circulation when the arteries are much relaxed, and pulmonary and systemic veins are overfilled with blood. In such cases it will tighten up the vessels, and, by augmenting the power of the systole, will force a large volume of blood into the arterial system. This accomplished, it should at once be stopped, and reliable remedies used. The use of nitroglycerin only partially relieves the damaging effects of digitalis and does not antidote its toxic action. [The routine administration of digitalis for any condition has long since been abandoned by progressive physicians, and the intelligent conception of the last author should command attention.]

Dionin.—Scherer[3] makes a plea for this morphin derivative (hydrochlorate of ethyl morphin) in the treatment of **bronchitis, tuberculosis, asthma, laryngitis,** and other affects of the respiratory passages. In doses of from ⅓ to ½ grain it exerts, according to Scherer, an excellent sedative influence, without causing unpleasant by-effects, as headache, nausea, constipation, etc. Compared with morphin and codein, he believes that dionin is milder and more harmless than the former, and stronger in action as well as more lasting in its effects than the latter. Kurtz[4] also speaks favorably of **dionin in pulmonary affections.** He finds, too, that its analgesic properties make it valuable in colicky and gastralgic pains, as well as in gastric cancer.

McAuliffe[5] states that a 5 % solution of **dionin instilled into the eye** every 5 minutes for half an hour gives relief in deep-seated inflammation beyond the reach of other analgesics. It causes some edema of the lids when it relieves the pain. Tuniewski[1] has found dionin very useful as an analgesic in iritis, pannus, and parenchymatous keratitis, but of no value in trachoma, blepharitis, acute conjunctivitis, or **glaucoma.** Schmitz[2] and Terson[3] also speak favorably of **dionin in ophthalmic practice.**

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[1] Med. News, Jan. 11, 1902.

[2] Med. News, May 3, 1902.

[3] Therap. Monats., Mar., 1902.

[4] Therap. der Gegenwart, May, 1902.

[5] Med. Critic, Feb., 1902.

Dormiol.—This compound is chemically dimethyl-ethyl-carbinol-chloral. It is produced by the action of chloral on amylene hydrate, and is a clear, transparent liquid, with a camphoraceous odor and a slight burning taste. It is miscible in all proportions with alcohol, ether, chloroform, and oils. In water it is but slowly soluble; it is, therefore, marketed in the form of a 50 % aqueous solution. The dose of the latter is from ½ to 1 teaspoonful, repeated in an hour, if necessary. Wederhake,[4] after experimenting with **dormiol** in numerous cases of **insomnia in the insane,** concludes that the drug is harmless, does not lose its efficiency even after prolonged administration, and does not cause any unpleasant after-effects. Its action is not so powerful as that of chloral, and it is therefore indicated in milder cases of insomnia. When pain is present, it acts only in combination with an opiate. It produces sleep within 15 to 30 minutes, and the effect lasts from 5 to 8 hours. According to Besançon and Dumont,[5] dormiol may with advantage replace chloral in cases of intractable **insomnia,** whether **of neurasthenic or hysteric origin,** or due to alcohol. They have never observed any disorder of circulation or respiration from its use. Furst,[6] Nola,[7] and Munk[8] also speak highly of **dormiol as a hypnotic and sedative.** [While the effects of dormiol are satisfactory for occasional use, like amylene hydrate, toleration is rapidly acquired.]

Emetin.—Maurel[9] concludes from a series of experiments on frogs that this drug is a powerful vasoconstrictor. In the congestive zone around a wound this action overcomes vasodilation and markedly reduces the degree of inflammation. This vasoconstrictor action, the author believes, explains the excellent results which have been obtained clinically by the employment of ipecac in affections accompanied by congestion, notably in the digestive and respiratory systems.

Eserin.—Von Noorden[10] has recently employed eserin in the treatment of intestinal atony. In 5 cases of tympanites, postoperative or following upon typhoid fever, salpingitis, etc., the author was able to bring about a complete disappearance of the distension by administering from $\frac{1}{40}$ to $\frac{1}{20}$ of a grain of eserin salicylate divided into 2, 3, or 4 doses. Owing to the rapid change of the drug in solution he suggests its employment in powder.

Ether.—Carter[11] has used a mixture of nitrous oxid and ether for producing anesthesia in 1000 cases. He says that the only danger connected with the use of nitrous oxid is asphyxia, hence the respiration and color of the patient must always be carefully watched. Anemic persons and children require but little of the drug. As a preliminary to ether, its advantage lies in its almost perfect safety, and its rapid and pleasant action. None of the prolonged and disagreeable sensations of ether inhalation are experienced. [A few observations of the author's method and practice seem to indicate that this, if not safer, is certainly a far more pleasant one for the patient.]

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[1] Die Heilkunde, VI, No. 2, 1902.

[2] Woch. f. Therap. u. Hyg. des Auges, IV, No. 38, 1902.

[3] Ophthal. Klinik, No. 17, 1901.

[4] Allg. Zeitschr. f. Psychiatrie, LVIII, 1901.

[5] Les Nouveaux Remèdes, Mar. 24, 1902.

[6] Deut. med Zeitschr., No. 91, 1901.

[7] Il Policlinico, VIII, 1902.

[8] Deut. Praxis, May, 1901.

[9] Comptes Soc. Biol., Oct. 25, 1901

[10] La Semaine Méd., Oct. 23, 1901.

[11] Med. Rec., Nov. 9, 1901.

Ethylene Bromid.—Scherbatscheff[1] has undertaken an investigation of **the direct and collateral effects of ethylene bromid and ethyl bromid,** because a case of fatal poisoning had occurred after the use of ethylene bromid by mistake. The patient, a woman, while on her way home, about an hour and a half after the operation, had been overcome with vomiting, had soon gone into collapse, and died the following morning. Experimental studies carried out on dogs and mice are then reported. Ethylene bromid produced in these animals symptoms similar to those noted in the case above mentioned. If the duration of the experiments was sufficiently long, and if the amount of ethylene bromid was large, death was produced. Narcosis was not caused in any of the animals. The respiration was increased in frequency, but was more superficial. The blood-pressure constantly sank. Death occurred partly from severe pulmonary congestion, partly from reduction of the blood-pressure, and possibly from some reduction in the alkalinity of the blood; although the latter was certainly not marked. Ethyl bromid produced no unfavorable collateral effects. Death occurred if the amount inhaled was great or if the inhalation was prolonged to beyond an hour, in many of the cases; but in some cases large amounts were taken, and observation was carried on over a long period, without any unfavorable effects being observed.

Eucain.—Cohn[2] calls attention to a new salt of eucain, **acetate of beta-eucain.** It is said to be superior to the hydrochlorate, in that it is freely soluble in water. In the normal eye it causes a little burning, rarely lasting more than half a minute, and the instillation is followed by a slight conjunctival hyperemia, in marked distinction to the blanching of the part when cocain is used. Commonly corneal anesthesia is produced in about 3 minutes. The drug, it is stated, does not influence the pupil, accommodation, nor the tension of the eye. Symptoms of intoxication were never noted.

Eucalyptus.—In a limited number of cases of diabetes, Faulde[3] has found that eucalyptus in the form of an infusion causes a marked lessening and even disappearance of the glycosuria. He observed that the effect of a fresh, warm infusion of the leaves was much more marked than that produced by the drug in any other form. This use of eucalyptus appears to be based solely upon empiricism. No explanation of the action of the drug is attempted.

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[1] Arch. f. exper. Path. u Pharm., Bd. XLVII. Hefte 1 u. 2, 1902.

[2] Die Med. Woche, Sept. 9, 1901.

[3] Glasgow Med. Jour., May, 1902.

Euchinin.—Mori[1] reports that **euchinin** in doses of from 4 to 8 grains a day **is capable of preventing malaria** in those not protected by living in mosquito-proof houses. It is advisable to begin treatment 4 or 5 months before the usual malarial season. He reports, further, that in a series of observations on 89 individuals under similar circumstances, in 42 who had been under treatment, only 5 contracted malaria; while in 47 not so treated 39 suffered. The 5 who contracted the disease had it in a mild form. Rocaz[2] has found euchinin fully as effective as quinin, while children take it more readily, since it has no bitter taste. It is also free from any deleterious action on the digestion.

Ferric Chlorid.—Patein[3] shows that perchlorid of iron gives excellent results in hemorrhages occurring in patients suffering from cancer of the uterus and in epistaxis, etc., provided that it is used at the temperature of 122° F. and in solutions of 1 : 20 or 1 : 50.

Formalin.—At a meeting of the British Congress on Tuberculosis, held in London in July, 1901, Maguire[4] reported **excellent results in tuberculosis from** intravenous injections of **formalin.** In a more recent note, Maguire[5] states that this treatment is still a trial, and as yet its results do not warrant any authoritative opinion, still less any positive statements as to its being a "cure for consumption." Fischer and Tieken[6] from experiments on guinea-pigs show that intraperitoneal **injections of dilute formalin** in infected guinea-pigs **have an unfavorable action** upon the tuberculous process. They conclude that the development of miliary tubercles, in spots that were actually bathed in dilute formalin, lead them to believe that it takes a much stronger solution of formalin to destroy tubercle bacilli than is commensurate with life.

Gelatin.—Miwa[7] quotes the works of a Chinese physician dating from 204 to 219 A. D., in which **gelatin is recommended as a hemostatic** for all kinds of hemorrhage. It has been used ever since for this purpose in China and for a thousand years in Japan. It was applied in aqueous solution or as a powder. Wood[8] has studied the **effect of the digestion of gelatin upon its styptic properties.** He concludes that pepsin digestion of gelatin does not destroy its coagulating effects on the blood; that the resulting product is dialyzable, and, therefore, capable of absorption; that the administration of gelatin by the mouth in the treatment of hemorrhage is, therefore, a rational procedure; and that gelatose seems to antagonize, if given in sufficient quantity, the anticoagulating action of peptone. Zibell[9] has undertaken to find out **upon what constituents the hemostatic qualities of gelatin depend.** As it has been proved that the addition of calcium salts to the blood increases its coagulability, he made careful quantitative estimations of the amount of calcium in 4 specimens of gelatin. He found that ordinarily gelatin contains about 0.6 % of calcium, so that 100 cc. of a 5 % solution would contain 0.03

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[1] Centralbl. f. Bakt., No. 20, p. 786, 1901.
[2] Gaz. Hebdom. de Bordeaux, No. 11, 1902.
[3] Bull. Gén. de Thérap., Aug. 15, 1901.
[4] See YEAR-BOOK for 1901.
[5] Brit. Med. Jour., Feb. 15, 1902.
[6] Trans. Chicago Path. Soc., Feb. 10, 1902.
[7] Centralbl. f. Chir., Mar. 1, 1902.
[8] Amer. Med., May 3, 1902.
[9] Münch. med. Woch., No. 42, 1901.

gram, or ½ grain. He believes, in the absence of a better explanation, that the activity of gelatin may be ascribed to the amount of calcium it contains. Holtschmidt[1] reports 5 cases of **melena neonatorum** occurring in the gynecologic clinic at Dresden during the year 1901, **in which subcutaneous injections of a 2 % solution of gelatin were used.** During the 7 years from 1894 to 1900, 7 of the 14 cases of melena occurring in this clinic resulted fatally. All of the 5 cases occurring in 1901 recovered. As a rule, 15 cc. of the gelatin solution was used, and in 2 instances only was this repeated. Döllner[2] also reports a successful result in a severe case of melena neonatorum in which he injected 40 cc. of a 2 % solution of gelatin in 4 doses in 5 hours. Thieme[3] reports satisfactory results in 12 cases of hemoptysis from the injection of 100 cc. of a 22 % solution of gelatin. Only one of the patients succumbed to the hemorrhage. Nieder[4] reports from von Leyden's clinic that he succeeded **in arresting a rebellious hemorrhage from the lungs,** which had lasted 8 days, **by the subcutaneous injection** of 60 cc. of a 2.5 % solution **of gelatin** in the right thorax, the site of the bleeding. The hemoptysis was arrested at once and did not recur. Hammelbacher[5] also reports favorably upon the use of gelatin in 4 rebellious cases of hemoptysis. Geraldini[6] reports 5 cases of severe hemorrhagic dysentery controlled by the subcutaneous or rectal injection of gelatin.

Attention has recently been drawn to the development of **tetanus after injections of gelatin.** Lorenz[7] reports 2 cases of tetanus resulting from the injection of 200 grams of a 1 % solution, and 100 grams of a 2 % solution, respectively. One patient died on the ninth day, the other within 24 hours. Krug,[8] who has previously reported a fatal case of tetanus following an injection of gelatin to arrest hemorrhage after extraction of a tooth, has been able to collect 5 other fatal cases and one in which the patient recovered. Gradenwitz[9] reports still another fatal case in a woman who had received a subcutaneous gelatin injection on account of profuse bleeding. Levy and Bruns[10] were able to produce **tetanus in guinea-pigs with** 4 out of 6 specimens of **commercial gelatin** used in the test. Schmiedicke[11] states that he has also found tetanus bacilli in commercial gelatin.

Sailer[12] describes the **technic** of the employment **of gelatin as a hemostatic** as follows: The gelatin is prepared as for ordinary mediums, using, however, 5 to 8 hundredths of a 1 % saline solution instead of bouillon. Since the degree of pain experienced on injection seems to be proportionate to the amount of turbidity, it is important thoroughly to clarify the solution with white of egg. Sterilization is then accomplished by heating the solution in an ordinary steam sterilizer 15 minutes for 3 successive days. In regard to the strength of the solution there seems to be considerable divergence of opinion. Generally, however, 5 % to

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[1] Münch. med. Woch., Jan. 7, 1902.
[2] Münch. med. Woch., May 27, 1902.
[3] Münch. med. Woch., Feb. 4, 1902.
[4] Deut. med. Woch., June 5, 1902.
[5] Münch. med. Woch., Dec. 10, 1902.
[6] Gaz. deg. Osped., No. 69, 1902.
[7] Deut. Zeitschr. f. Chir., Nov., 1901.
[8] Therap. Monats., June, 1902.
[9] Centralbl. f. Gynäk., Sept. 13, 1902.
[10] Deut. med. Woch., Feb. 20, 1902.
[11] Deut. med. Woch., Mar. 13, 1902.
[12] Therap. Gaz., Aug., 1901.

10 % solutions are employed locally, and 1 % or 2 % for subcutaneous injection. About 10 ounces of the 1 % solution are required. The preferred situations for injections are between the shoulder-blades, under the breast, and on the outer side of the thigh. They are indicated in all hemorrhages, such as hemoptysis, hematemesis, metrorrhagia, melena neonatorum, purpura, and the purpuric forms of the infectious diseases, and are contraindicated in only one condition, viz., acute nephritis.

Glutol.—This preparation is made by exposing gelatin to the vapor of formalin. By contact with living tissues the gelatin is dissolved and formalin liberated. Moir[1] recommends a mixture of **glutol and soft paraffin** (1 to 4 or 1 to 8) **as a preventive of severe pitting in smallpox.** Moir,[2] Lobb,[3] and Maguire[4] speaks favorably of **glutol as an antiseptic dressing for wounds.**

Gluton.—Brat[5] reports on a new dietetic preparation known as gluton. It is a yellowish powder, belongs to the class of glutoses, and is prepared by the action of acids on gelatin at a high temperature for several hours. The acids are neutralized, and the dialyzed and filtered product is evaporated to dryness and reduced to powder. It is added to soups, bouillon, and other liquid preparations, the claim being made that it is very nutritious and easily digested. It is readily soluble in water and does not gelatinize even in concentration solutions.

Guaiacol.—Rachford[6] calls attention to the great value of **guaiacol in the treatment of tuberculosis in children.** Guaiacol, in his opinion, far outclasses all other drugs in the treatment of this condition. He recommends a teaspoonful of the following ointment to be rubbed into the chest each day at bedtime: Guaiacol, 1 dram;. lanolin, 2 drams; lard, 4 drams. He has used this combination for the past 8 years in almost every case of tuberculosis in infancy and childhood which he has had an opportunity to treat, and his experience in many hundreds of cases has convinced him of its value. He has found it particularly efficacious in lymphatic and in peritoneal tuberculosis. He claims that in the latter disease good results are at once apparent, and that the patient, as a rule, slowly but steadily recovers.

Bocchi[7] reports 10 cases of **acute gonorrheal epididymitis and orchitis treated with guaiacol** in a 10 % vaselin salve. The pain is relieved almost immediately and disappears permanently after 4 or 5 days. The simplicity, convenience, and efficacy of this method of treatment, render it superior to all other methods in the author's experience.

Grande[8] reports 12 cases of epidemic **parotiditis** in which he used a 5 % ointment of guaiacol, rubbing it over the entire parotid region and wrapping the part in cotton. This application was renewed after 24 hours, and a third time if necessary. He states that guaiacol is the most efficient remedy that has come to his notice in the therapy of mumps.

Guaiacol Benzoate.—Earp[9] prefers the benzoate to other preparations of guaiacol, as it is comparatively tasteless and odorless, and does not disturb digestion. In doses of from 3 to 6 grains, 4 times a day, it proved very useful in his hands in phthisis, catarrhal conditions of the intestines, and irritable states of the bladder.

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[1] Therapist, Feb. 5, 1902.
[2] Therapist, July 15, 1901.
[3] Therapist, Aug. 15, 1901.
[4] Ibid.
[5] Deut. med. Woch., Jan. 9, 1902.
[6] Arch. of Ped., Dec., 1901.
[7] Gaz. degli Osped., Mar. 23, 1902.
[8] Gaz. degli Osped., Aug. 10, 1902.
[9] Cincinnati Lancet-Clinic, Feb., 1902.

Guaiacum.—Schaer[1] concludes that the therapeutic properties of guaiac depend solely upon the saponin, which is most abundant in the bark, less so in the outer part of the wood, still less in the medulla, and least of all in the resin. The fact that the decoction of the wood contains the most saponin of all galenical preparations, and has been found to be the most active preparation, seems to support this theory, as do also the recent observations that other ingredients, such as guaiaconic acid, have no effect in syphilis.

Hedonal.—Thaly[2] has employed hedonal (the ester of methyl-propyl-carbinol-carbamic acid) as an hypnotic in doses of from 15 to 30 grains, occasionally 45 grains, divided into 2 portions and given 2 hours before retiring. His observations concur closely with the numerous other reports that have been published on hedonal [see YEAR-BOOK for 1902], which demonstrate that, while the drug is less powerful than the hypnotics in common use, it is a safe remedy and particularly adapted for the treatment of the milder forms of insomnia in nervous, neurasthenic, or hysteric subjects.

Heroin.—Marshall[3] compares the action of this drug with that of dionin and morphin. He states that both heroin and dionin [cf. Dionin: *v. supra*] differ from morphin in their action. In both, the convulsive effect is increased at the expense of the sedative one. When administered to lower animals, these drugs produce transient depression, followed by convulsions. These convulsions closely simulate those which are produced by the action of drugs on the medulla, and in part at least the action of dionin, and especially of heroin, is due to such an effect. Of the two drugs, dionin approximates more closely than heroin to morphin in action. It is more sedative than heroin, less convulsant, less active to the respiratory center, and also less toxic. Compared with morphin, it is less toxic, less sedative, and most convulsant, and less depressant to the respiratory center. Heroin, compared to morphin, is much more toxic, much more convulsant, and much more active to the respiratory center. From experiments on animals, Marshall concludes that in appropriate doses heroin can prolong inspiration and increase the depth of respiration, but that the limits of its beneficial action, unless simple slowing is required, are soon passed. Slowing is always produced. In moderate doses it depresses the respiratory center. Dionin appears to be slightly more sedative than codein, but otherwise it possesses the same action. Heroin has a much greater effect on respiration, but he is doubtful if this action will prove of much use therapeutically. The limits of its beneficial action are too narrow.

Elischer[4] has made some experiments with **heroin hydrochlorid,** with the view of testing its local analgesic action. The drug was introduced some time ago by Mirtl into gynecology as an application in painful affections of the genital tract. Elischer employed it for this purpose on glycerin tampons in the strength of 1 : 1000, introducing about $\frac{1}{8}$ to $\frac{1}{4}$ grain into the vagina at each application, and allowing the tampon to remain several hours. He confirms the analgesic effect of the drug and regards it as an extremely useful auxiliary in the treatment of incurable uterine cancer.

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[1] Arch. f. exper. Path. u. Pharm., Bd. XLVII, 1902.

[2] Pester med. chir. Presse, No. 41, 1901.

[3] Brit. med. Jour., Oct. 18, 1902.

[4] Die Heilkunde, Feb., 1902.

Hydrastin Hydrochlorate.—Saenger[1] has found that hydrastin hydrochlorate has a surprisingly powerful effect in loosening tenacious masses and favoring their expectoration in chronic bronchitis. It is a stable drug and he recommends it urgently. He prescribes for adults 10 to 20 drops of a solution of 15 grains in 5 drams of water, 3 or 4 times a day.

Hydrochloric Acid.—Soupault[2] calls attention to the value of hydrochloric acid in those forms of chronic diarrhea which are independent of anatomic lesions and of local or general intoxications. The stools are fluid and more or less frequent, with occasional colic. The condition may last for months or years and is not relieved by ordinary treatment. Soupault, however, has found that the disease is quickly and durably cured by the administration of hydrochloric acid, beginning with small doses and gradually increasing the amount to 1 or 2 grams a day. Out of 38 cases thus treated, 23 were completely cured, and the others were greatly relieved. Mathieu[3] corroborates Soupault's observations concerning the efficacy of hydrochloric acid in the treatment of chronic diarrhea.

Hydrogen Dioxid.—Roger[4] has witnessed remarkably prompt cures of **dysenteriform colitis** from 1 to 3 rectal injections daily of a pint or quart of dilute hydrogen dioxid (hydrogen dioxid, 100 cc., sodium chlorid, 5 gm., sodium phosphate, 3 gm., sodium bicarbonate, 0.5 gm., and water, 900 cc.). Roger states that these injections also give good results in **mucomembranous colitis,** although less striking than in the other variety. This method of treatment has also been used by Rocaz[5] with great benefit in 10 cases of **dysentery occurring in children** from 2 to 12 years of age. That a cure may be assured, it is necessary, according to Rocaz, to continue the medication for some days after the subsidence of the symptoms.

Hyoscin.—Robin[6] has been impressed with the great benefit derived in 5 cases of **tremor** from the subcutaneous injection of hyoscin. The tremor was of senile origin or due to Parkinson's disease, or to chorea, and improvement was pronounced in all but one. He commenced with $\frac{1}{64}$ grain and increased gradually to $\frac{1}{15}$ grain a day. One of the patients took $\frac{1}{25}$ grain a day for 19 days, and although not entirely cured was so much improved that he was able to resume his occupation. Bury[7] is also of the opinion that hyoscin is the most useful drug in **paralysis agitans,** but believes that it should be used with great care. Hare[1] states that he has employed the method recommended by Lott[2] of treating various forms of **drug-habit** with massive doses of hyoscin in 6 cases with extraordinary results from the following points of view: First, the patient can take massive doses for days at a time, as much as ¼ grain each day hypodermatically, with no evil effects on any vital function; second, they suffer very slightly, if at all, from the immediate withdrawal of morphin; and, third, and more surprising, the desire for the drug is largely, if not entirely, dissipated after a few days. Four cases are reported in detail. Bering[3] also has used large doses of hyoscin ($\frac{1}{100}$ grain hourly, day and night) with very good results in a case of acute alcoholism. [The point hitherto overlooked seems to have been that the doses should be massive, yet discretion must be exercised, for toxic effects are often, even under these conditions, met with.]

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[1] Wien. klin. Rundschau, No. 20, 1902.
[2] Bull. Gén. de Therap., No. 12, 1902.
[3] Bull. Gén. de Therap., No. 14, 1902.
[4] Presse Méd., Jan. 1, 1902.
[5] Jour. de Méd. Bordeaux, Dec., 1901.
[6] Bull. Gén. de Therap., No. 7, 1902.
[7] Lancet, No. 4103, 1902.

According to Baker,[4] hyoscin hydrobromate comes nearer being the ideal **cycloplegic** than any other drug in our possession. It is safe and sure; with the possible exception of homatropin, its effects are the shortest in duration; it is the most prompt in its action; its exhibition consumes the least possible time, as only 1 drop of the solution is required to produce cycloplegia, and its solutions are remarkably stable. The author uses 0.5 % solution, of which, as mentioned, 1 drop is instilled. He has used it in upward of 3000 cases for refracting, and has observed no bad effects.

Ichthargan.—Saalfeld[5] has used this remedy (a combination of silver with ichthyol-sulfonic acid, containing 30 % of silver) in 140 cases of **gonorrhea.** In acute cases injections containing from 1 grain to 1½ grains in 8 ounces were ordered every 3 hours. Irrigations of the urethra were performed with solutions of 1 : 4000 up to 1 : 750. For instillation, solutions of 0.5 % to 5 % were used. The author believes that ichthargan is less irritating than the older silver preparation and the gonorrhea can be cured by using this drug only, graduating the solutions according to the stage and intensity of the disease. Goldberg[6] also reports favorable results from ichthargan in 60 cases of gonorrhea.

Ichthyol.—Mostkoff[7] has employed ichthyol in the treatment of 50 cases of **pulmonary tuberculosis.** He administered it with an equal quantity of water, in doses of from 5 to 20 drops, thrice daily, after meals, using as a vehicle wine or black coffee. Symptoms of intolerance were not observed; on the contrary, it is stated that the appetite improved and the patients gained in weight and strength. Spangler[8] and de Renzi[9] also speak favorably of ichthyol in the treatment of tuberculosis.

Iodipin.—This preparation, an addition-product of iodin and oil of sesame, containing 10 % or 25 % of iodin, has been used quite extensively during the past year as a **substitute for iodin.** According to Blanck,[1] the drug passes the stomach unaltered and is partly split up in the intestines. The greater part, however, it is claimed, is absorbed and deposited as an iodin-fat in the tissues, whence the iodin is slowly liberated. A gradual and prolonged action of the drug is in this manner assured. The same circumstances, the author believes, account for the absence or mildness of untoward effects. The drug can be administered by the mouth, by the rectum, by inunction, or subcutaneously. By the mouth, the dose is from 1 to 4 drams or more of the 10 % solution; for hypodermic injection, 1 to 2 drams or more of the 25 % solution. Hoenigschmied[2] has used iodipin with success in tuberculosis of the glands and bones, in impetigo, sciatica, and emphysema. Friedländer,[3] Wanke,[4] Holzhauser,[5] and Rille[6] speak favorably of its action in syphilis. Kreibich[7] administered the 25 % solution subcutaneously with excellent results in a case of actinomycosis of the cheek. Von Hymmen[8] has observed beneficial effects from iodipin in a variety of ocular affections.

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[1] Med. News, June 7, 1902.
[2] Therap. Gaz., Aug. 15, 1902.
[3] Therap. Monats., Mar., 1902.
[4] Therap. Gaz., Jan., 1902.
[5] Jour. Am. Med. Assoc., May 3, 1902.
[6] Therap. Monats., Mar., 1902.
[7] Allg. med. Central-Zeit., No. 76, 1902.
[8] Proc. Phila. Co. Med. Soc., Jan., 1902.
[9] Berl. klin. Woch., May 5, 1902.

Iodin-phosphor.—This substance contains $\frac{1}{50}$ grain of phosphorus in 10 minims of iodipin of 25 % strength. Bartholow[9] states that when it is given hypodermatically in doses of 20 minims it promotes in a marked degree nutrition, increases the body-weight, and removes or modifies the symptoms of various nervous affections not readily amenable to treatment, including spinal sclerosis, neuralgia, neurasthenia, gout, chronic rheumatism, etc.

Iodo-mercury Cacodylate.—Civatte and Fraisse[10] have tested this preparation in 60 cases of syphilis. As the results were so superior to mercurial treatment alone, they conclude that the addition of sodium cacodylate is indicated whenever the patient is depressed, neurasthenic, or emaciated. The dose is 1 cc., increased to 2 cc., hypodermatically.

Izal.—This is a proprietary cresol preparation recently discovered in the investigation of the by-products formed during the conversion of coal into coke. Tunnicliffe[11] believes that izal is an efficient and cheap substitute for guaiacol carbonate in the treatment of **phthisis.** He has found it very beneficial in active tuberculosis with abundant fetid sputum and in cases with diarrhea due either to fermentation or to intestinal tuberculosis. In cases in which the bronchitic element was pronounced it was less useful. Gordon[12] concludes from a study of the effects of izal on **intestinal fermentation** that doses which exert no unfavorable symptoms diminish the number of microorganisms in the feces to a very striking extent, and that the drug appears to be more active as an intestinal disinfectant than most substances proposed for this purpose.

Lecithin.—French authorities continue to speak favorably of this preparation of glycero-phosphates. It is usually given in capsules, in doses of from 2 to 4 grains, thrice daily, after meals. Linn[1] has found it very efficacious in neurasthenia, diabetes, and tuberculosis. Huchard[2] and Lancereaux[3] each report cases of diabetes in which they prescribed lecithin with excellent results. Claude and Zaky[4] conclude from a series of clinical studies that lecithin is a valuable adjuvant to other therapeutic measures in tuberculosis.

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[1] Die med. Woch., Dec., 1901.

[2] Aerzt. Zeit., No. 28, 1901.

[3] Allg. med. Central-Zeit., No. 58, 1901.

[4] Correspon. Blätter, Nos. 6 and 7, 1901.

[5] Therap. Monats., No. 8, 1901.

[6] Wien. klin. Woch., No. 30, 1901.

[7] Wien. klin. Woch., No. 4, 1902.

[8] Ophthal. Klinik, No. 24, 1901.

[9] Amer. Med., July 26, 1902.

[10] Annal de Derm., Oct. 1, 1902.

[11] Brit. Med. Jour., Jan. 18, 1902.

[12] Lancet, No. 4097, 1902.

Lycetol.—Vicario[5] has recently published an account of his researches on the relative solubilities of the various salts formed by the combination of uric acid with such substances as are employed for its removal from the human body. He finds that at the normal temperature of the body the acid urate of lithium is 1½ times more soluble than the acid urate of sodium; that the urates of urotropin and of piperazin are 8 times more soluble than lithium urate, and that lysidin urate is 20 times, and lycetol (dimethyl piperazin) urate 23 times, as soluble as lithium urate.

Lysol.—Burgl[6] has added 2 cases of **poisoning with lysol** to the 16 cases already published. The first, a male infant, 5 days old, received a coffee-spoonful of lysol in mistake for syrup of rhubarb. Death resulted in 14½ hours. The second case was that of a girl, aged 8 years, who received a teaspoonful of lysol in mistake for a dram of infusion of ipecac. Death resulted within a few hours. Of the 18 cases, 13 were poisoned by internal administration, and 6 of these died, while the remaining 5 were poisoned by external application (3 times undiluted, once in 1 % solution used in large quantities for washing out the uterus, and once the strength is not mentioned), with 3 deaths.

Mannitol Pentanitrate.—Marshall and Wigner[7] conclude from a study of the vasodilator action of this drug that it is less active than erythrol tetranitrate, but much more active than mannitol hexanitrate. Its only great advantage over erythrol tetranitrate is its relative cheapness when prepared on a large scale. It can, however, in double the dose produce all the effects of erythrol tetranitrate. It is also somewhat more stable than the latter, and more soluble in oil of theobroma, with which it must be mixed to prevent explosion. It possesses the usual ill-effects of organic nitrates—slight heaviness and tendency to headache—and it also has a bitter taste.

Mercury.—Werler[8] lauds the use of colloidal mercury as the most effectual and convenient method of antisyphilitic treatment for the general practitioner. A 10 % salve of this soluble mercury is absorbed through the skin far more rapidly and thoroughly, it is stated, than other preparations of mercury in vogue. It is mild, nonirritating, and nontoxic. The author prefers it even for internal administration. The mercury in this form is eliminated very slowly, regularly, and gradually.

Mercury Lactate.—Gaucher[9] asserts that the neutral lactate of mercury is of all mercurial salts the one best adapted for the **treatment of syphilis,** either by the mouth or by subcutaneous injection. It is

[1] Jour. des Praticiens, June 22, 1901.

[2] Jour. des Praticiens, July 13, 1901.

[3] Bull. Gén. de Thérap., July 15, 1901.

[4] La Presse Méd., Sept. 28, 1901.

[5] Les Nouveaux Remèdes, Mar. 24, 1902.

[6] Münch. med. Woch., Sept. 24, 1901.

[7] Brit. Med. Jour., Oct. 18, 1902.

[8] Therap. Monats., April, 1902.

[9] Bull. Soc. Méd. Hôp de Paris, Mar. 6, 1902.

scarcely at all irritating and contains so much mercury that $\frac{1}{8}$ grain a day subcutaneously is an ample dose.

Mercury Succinimide.—Horovitz[1] strongly recommends this salt of mercury for the treatment of syphilis. He employs it hypodermatically in the following solution: Mercury succininide, 5; cocain hydrochlorate, 2; distilled water, 100.

Methylene-blue.—De Blasi[2] administered methylene-blue in 100 cases of **malaria** with complete cure in 62. He found that the drug was effective only when the parasite had been circulating for some time in the blood. It seemed to have a tendency in pregnant patients to induce contractions of the uterus, and he therefore warns against its use during the last 2 months of pregnancy. He gave up to 30 grains in the 24 hours, after eating. Iwanoff[3] concludes from a study of the action of methylene-blue in malarial fever that the drug has little effect upon the younger parasites, while it markedly affects the mature forms, its action being the reverse of that of quinin.

Hughes and Lovelace[4] have used **methylene-blue as a sedative in** 22 cases of **insanity** with wild excitement. In only 6 out of the 22 cases did it fail to calm the patient. The sedative effect was noticeable in from 3 to 4 hours after the administration of the drug, lasted for from 15 to 20 hours, and seemed more like natural quietude than the dulness induced by other sedative remedies. No depression was observed, except in one case. The patients slept well at night, but during the day no hypnotic effect could be produced. Unpleasant symptoms were rarely noticed, and could be attributed to prolonged use of the drug. The remedy can be given by the mouth or hypodermatically, 2 grains and 1 grain being the average respective doses, repeated 2 or 3 times daily and, if necessary, continued for days and even weeks.

Nargol.—Schwarz[5] has employed nargol (a combination of nucleinic acid and silver, containing 10 % of silver) extensively and made clinical comparisons with protargol in 200 cases, nargol being used in one eye and protargol in the other under as nearly the same conditions as possible. Schwarz concludes that nargol is relatively nonirritating, has considerable range as an astringent, as well as superior penetrating power, stability, and solubility, and is most efficient in 10 % to 20 % solutions, which should not be kept more than 5 weeks. In solutions of equal strength nargol is more stable and less irritating than protargol, and appears to be equally efficient.

Olive Oil.—Thomson[6] believes that scarcely 1 out of 20 of those who suffer from gall-stones will ever need to undergo operation. He regards olive oil properly administered as the most efficient remedy, but holds that the old idea of its action is wrong. According to Thomson, it acts by exciting a watery flow from mucous membranes. He advises patients with cholelithiasis to take an ounce or two for 10 consecutive nights, then to intermit it for about a week to avoid gastric disturbance, and then to resume it again for 10 more nights. The result is said to be not immediate relief, but progressive amelioration of symptoms with the attacks becoming fewer and lighter until they cease permanently.

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[1] Centralbl. f. die Gesammte Therap., vol. xix, 1901.

[2] Gaz. degli Osped., Mar. 23, 1902.

[3] Deut. med. Woch., No. 18, 1901.

[4] Phila. Med. Jour., Mar. 22, 1902.

[5] Medicine, Mar, 1902.

[6] N. Y. Med. Jour., April 19, 1902.

Pankreon.—This is described as a preparation obtained by the action of tannic acid on pancreatin. It is a grayish, odorless powder, the taste of which is not unpleasant. Fifteen grains of pankreon at a temperature of 40° C. (104° F.) are capable of digesting 83 % of 1500 grains of egg-albumen in 15 minutes. The advantage claimed for this new preparation is its resistance to hydrochloric acid, by which ordinary pancreatin is destroyed in about 1½ hours. According to Hemmeter,[1] pankreon is of advantage in cases of achylia gastrica, chronic atrophic gastritis, and intestinal atrophy. It should be given a trial when examination of the stools shows that a considerable portion of the ingesta passes through undigested.

Papain.—Mendel[2] concludes from a study of the action of the vegetable proteolytic enzymes that papain, papayotin, or caroid, as it is variously called, an enzyme obtained from the melon tree (*Carica papaya*), apparently differs somewhat from the two proteolytic enzymes, pepsin and trypsin. While the products of the papain digestion of proteids resemble quite closely those of pepsin, so far as they have been examined in detail, the enzyme differs from ordinary animal pepsin in that it acts readily in both neutral and alkaline mediums. On the other hand, although papain is comparable with trypsin in exerting a solvent action in fluids of various reactions, the failure to form leucin, tyrosin, or tryptophan in appreciable quantities places it in a class of its own for the present.

Paraldehyd.—Brissemort[3] calls attention to the incompatibility of a combination of potassium bromid and paraldehyd. He states that the oxidizing properties of the latter convert the bromid into a bromate, thus weakening the therapeutic values of both drugs.

Pepsin.—Hemmeter[4] believes that there is rarely any indication for the use of pepsin, for whenever a test-meal shows free HCl, pepsin of necessity must be present in sufficient amounts; and even when HCl is absent, pepsin or pepsinogen is, as a rule, still present. Assuming a case in which the last vestige of even pepsinogen secretion has been lost, the introduction of the ferment might be of utility, but the enormous quantities of HCl necessary to bring about proper action of the pepsin could not be tolerated by any diseased stomach. And, again, in cases in which pepsinogen is still formed, the addition of HCl simply will suffice to convert it into a complete ferment.

Thibault[5] has studied the **effect of alcohol upon the activity of pepsin,** varying the strength of the alcohol and the duration of the experiments. He finds that, provided the alcoholic strength is below 12.5 %, the solution shows no immediate loss of strength, but above this strength the solution immediately undergoes deterioration. The peptonizing power of the solution is almost destroyed at the end of 4 months, whether the alcoholic strength be above or below 12.5 %. Hence it appears that pepsin should not be prescribed in alcoholic mediums, as the ferment is destroyed to a greater or less extent according to the age of the liquid.

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[1] Med. News, June 7, 1902.
[2] Am. Jour. Med. Sci., Aug., 1902.
[3] Jour. des Praticiens, Jan. 18, 1902.
[4] Med. News, June 7, 1902.
[5] Jour. de Pharm. et de chim., Feb 15, 1902.

Petroleum.—Reyburn[1] states that if we consider the entire insolubility of petrolatum and its compounds in either the gastric juice or the fluid of the intestinal canal, and the fact that when petrolatum is given internally the whole of it can be recovered from the feces, we are warranted in assuming that petrolatum can in no sense be considered a substitute for cod-liver oil as a nutrient and restorative, and that whatever usefulness it may have as a remedy must depend upon its lubricating and demulcent properties. [This conclusion is based on careful observations and should be considered final.]

Phosphorus.—Leo[2] agrees with Monti [see YEAR-BOOK for 1902] that no conclusive proof has yet been offered of a specific action of phosphorus in rachitis. Leo relies principally on dietetic and hygienic measures, especially the systematic administration of olive oil. If no improvement follows this treatment, he gives phosphorus in small doses, not over 10 drops of a 0.01 % solution.

Phototherapy.—Bang[3] states that out of 640 cases of **lupus** treated by sunlight or electric light after the method of Finsen only 11 did not show much improvement. Absolute cures resulted in 456 cases, 130 of which had not had recurrence while under observation. The cicatrices, according to Bang, are small, and there is no pain, but the applications must be continued for a long time, and the apparatus is expensive. Gaston,[4] Leredde,[5] and Veliaminoff[6] also make favorable reports on the light treatment of lupus vulgaris and lupus erythematosus.

Picric Acid.—MacLennan[7] recommends a saturated solution of picric acid as a local remedy in the treatment of acute eczema and erysipelas. In erysipelas, he finds that it stops the spread of the morbid process, and relieves the pain and burning better than carbolic acid, inert powders, or ichthyol. Fothergill[8] also speaks favorably of a saturated solution of picric acid in erysipelas.

Pituitary Body.—Golla[9] finds that when the pressor substance of the pituitary body is injected subcutaneously it slows the heart by prolonging systole, raises the blood-pressure, and excites diuresis. When given by the mouth, these results did not follow. He concludes that the cardiac, vascular, and diuretic action of the pressor substance indicates that it may be of service in cases of heart disease. Kuh[10] reviews the treatment of akromegaly with pituitary bodies and reports 3 cases of his own in which the drug was used. One of these received little, if any, benefit from the treatment, while in the other two cases, for 1½ and 1 year respectively, the condition of the patients seemed decidedly improved.

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[1] Med. News, Aug. 24, 1902.

[2] Therap. der Gegenwart, Dec., 1901.

[3] Berl. klin. Woch., Dec. 2, 1901.

[4] Ann. de Derm., April, 1902.

[5] Ibid.

[6] Vratch, Jan. 19, 1902.

[7] Montreal Méd., Aug., 1901.

[8] Brit. Med. Jour., June 28, 1902.

[9] Lancet, Feb. 15, 1902.

[10] Jour. Am. Med. Assoc., Feb. 1, 1902.

Potassium Iodid.—Lesser[1] calls attention to the irritant effects of calomel, applied locally or ingested, while the patient is under the influence of potassium iodid. The iodin ingested in the form of potassium iodid is eliminated in the secretion of every mucous membrane, and when it comes in contact with calomel, the caustic compound (mercury iodid) that results is liable to form ulcers, in the depths of which yellow crystals of the mercury iodid are plainly visible. Rabbits treated with potassium iodid and then receiving a stomach injection of calomel exhibited numerous ulcerations in the gastric mucosa. As potassium iodid passes into the milk, calomel should not be prescribed for nurslings when the mother is or has been taking potassium iodid. Calomel, mercurous iodid, and mercuric iodid all share this caustic property, while the salicylate, thymol-acetate, sublimate, and gray oil may be administered with impunity when the patient is under the influence of potassium iodid. Stockman and Charteris[2] conclude from an experimental study of the action of iodids on the heart and circulation that potassium or sodium iodid in therapeutic doses does not modify the physical conditions of the circulation, and that therefore they neither directly weaken the heart nor dilate the arterioles. Carter,[3] in 4 clinical observations made by means of the sphygmometer, could detect no effect upon the blood-pressure from therapeutic doses of potassium iodid (10 grains thrice daily). Stengel[4] calls attention to the development of **purpura in chronic iodism.** In several cases there were marked fetor of the breath, soreness and sponginess of the gums, tremors, sweating, rapid heart, enlargement of the thyroid gland, and extensive purpura, especially on the legs.

Purgen.—Purgen, or phenolphthalein, is a synthetic compound belonging to a class of bodies known as phthaleins, which may be regarded as derivatives of tri-phenol-methane. Like purgatin or anthrapurpurin diacetate, it is related to **anthraquinone**, the purgative principle contained in aloes, rhubarb, senna, etc. According to Tunnicliffe,[5] purgatin has several drawbacks: it often causes considerable colic; it colors the urine a bright red; and it seems occasionally to cause some irritation of the kidneys. From a study of the therapeutic action of the new compound, purgen, he draws the following conclusions: For children, purgen in doses of from ¾ to 2¼ grains is a useful aperient; for ordinary adults it must be given in doses of from 1½ to 4½ grains; in cases of obstinate constipation the dose must be increased to 15 grains; it produces purgation in jaundice; it has no irritant action on the kidneys; and its depressant action on the circulation is less than that of magnesium sulfate.

Pyridin.—Méry[6] states that the most efficacious method of treating **asthmatic paroxysms in children** is with pyridin. He recommends the inhalation of from 7 to 8 drops of the substance from a saucer or handkerchief, 4 or 5 times a day. Cacchiole[7] has obtained good results from inhalations of pyridin in **pertussis.** In connection with the inhalations he recommends the administration of bromids. [We would class the inhalation of pyridin in pertussis as among the disciplinary measures formerly in vogue.]

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[1] Deut med. Woch., Nov. 28, 1902.

[2] Brit. Med. Jour., Nov. 23, 1901.

[3] Am. Jour. Med. Sci., Dec., 1901.

[4] Therap. Gaz., Jan., 1902.

[5] Brit. Med. Jour., Oct. 18, 1902.

[6] Gaz. des Mal. Enf., Feb. 13, 1902.

[7] Gaz. deg. Osped. e del. clin., Aug 10. 1902.

Quinin Hydrobromate.—Ferguson,[1] after experimenting with the various salts of quinin for hypodermatic use, concludes that the bihydrobromate, which is soluble in 6 parts of distilled water, is the best. He has found this salt perfectly stable, and more reliable and less irritating than other forms of quinin. He uses it by injecting 3 grains of the drug dissolved in 20 minims of water under the skin of the arm, thigh, or abdomen, on alternate days. He keeps a syringe solely for this purpose, and disinfects the syringe, the patient's skin, and his own hands, and boils the solution before every injection. He thinks that very few patients with malaria can resist 6 doses of 3 grains each of quinin bihydrobromate.

Sclavo's Serum.—Cigognani[2] reports 14 cases of **malignant pustule** cured by Sclavo's serum, and reaches the following conclusions: The serum, even when administered in large doses, always proved harmless; a few hours after the injections a peculiar euphoria was noticed; small injections (10 cc. to 20 cc.) were more efficacious than large ones (40 cc.) injected at one time; injections into the veins were more effectual and yielded quicker results than subcutaneous injections; pustules treated with injections healed very quickly. Even serious cases were cured with the serum in which otherwise the prognosis would have been very grave.

Silver.—Wenckebach[3] reports a case of **endocarditis,** apparently of a septic type, in which beneficial results followed intravenous injections of from $\frac{1}{5}$ to $\frac{3}{4}$ grain of soluble silver in the form of a 1 % or 2 % solution. Woyer[4] reports 3 cases of **puerperal sepsis** in which rapid improvement followed inunctions of soluble silver, 45 grains at intervals of 10 hours. Toff[5] also speaks favorably of the action of soluble silver (unguentum Credé) in 5 cases of sepsis.

Sodium Cacodylate.—[See also Arrhenal.] Mendel[6] states that he has used this remedy with great satisfaction in 30 cases, including cases of diabetes, tuberculosis, chlorosis, migraine, sciatica, Graves's disease, and hysteria. He prefers the intravenous method of administration, and employs the drug in doses of $\frac{2}{3}$ grain, gradually increased to 2 grains. Fraser,[7] believing that an inert compound of arsenic could not produce the therapeutic effects of the more active arsenic compounds, made a series of experiments with sodium cacodylate in cases of chorea, eczema, and leukemia. He failed to find that the cacodylate produced the extraordinary effects which were claimed for it. He points out that the arsenic is so firmly locked up with the methyl groups in the cacodylates that the salts give only the reactions of the base and not of the arsenic, and in order to liberate the arsenic prolonged oxidation with mineral acids is necessary. He examined the urine of patients who were taking cacodylates and found that it gave no reaction with Marsh's test. He concludes, therefore, that the cacodylates pass through the body in a state of stable combination, with the arsenic so firmly locked up that it cannot be disassociated to form active substances, and thus exert the toxic effects of the usually employed preparations of arsenic. Fraser believes that hypodermatic injections of cacodylates can be given until an equivalent of liquor arsenicalis is administered, but that these preparations are very feeble. He finds that even when administered by the mouth in therapeutic doses they are likely to cause gastric or intestinal disturbances, and to impart a strong garlicky odor to the breath, which is repugnant to most patients.

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[1] Brit. Med. Jour., Feb. 22, 1902.

[2] Gaz. degli Osped., No. 114, 1901.

[3] Therap. d Gegenwart, No. 2, 1902.

[4] Münch. med. Woch., No. 42, 1902

[5] Münch. med. Woch., No. 17, 1902.

[6] Therap. Monats., April, 1902.

[7] Med. Presse, Mar. 12, 1902.

Sodium Nitrate.—Binz and Gerlinger[1] prove experimentally that sodium nitrate is partially converted in the body into sodium nitrite. In all the animals tested appreciable amounts of nitrites were found in the urine, and the presence of methemoglobin in the blood was manifest by its brown color and the grayish tinge of the mucous membranes. Owing to the small therapeutic doses of the nitrate, however, little need be feared from its reduction in man.

Sodium Nitrite.—Brunton[2] has found a combination of potassium bicarbonate 28 grains, potassium nitrate 18 grains, and sodium nitrite $\frac{1}{20}$ grain, taken every morning in a large glass of water, very efficacious in cases of **high arterial tension of gouty origin.** He believes that if the treatment be instituted in time, while a much accentuated aortic sound is as yet the only symptom, angina pectoris and cerebral hemorrhage may be warded off for an indefinite period. Carter[3] concludes from a series of clinical experiments, in which the sphygmometer was used to gage the blood-pressure, that the most satisfactory drug for lowering blood-pressure, whether increase is due to nephritis or marked arteriosclerosis, is sodium nitrite, given in doses of from 2 to 3 grains every 2 to 4 hours. He asserts that it acts in about 26 minutes, and that the effect lasts on the average 1 hour and 44 minutes. [Pure sodium nitrite is rarely met with in the shops.]

Sodium Salicylate.—Snell[4] reports a case of complete blindness following the injection of between 140 and 160 grains of sodium salicylate in divided doses. The patient was a young woman suffering from acute rheumatism. Death resulted on the third day after the development of the amaurosis from endocarditis. Ophthalmoscopic examination showed nothing pathologic.

Solanum Carolinense.—Thrush[5] draws the following conclusions from the use of this drug in 25 cases of **epilepsy**: It is of great value (probably better than any one known remedy) in grand mal of idiopathic type without hereditary taint and when the disease has begun beyond the age of childhood. It is perhaps next of greatest value in hystero-epilepsy. In petit mal it is rarely of much service. The fluid extract made freshly is the best form of pharmaceutic preparation, and should be given in ascending doses commencing with 1 fluidram and increasing to the full constitutional effect. The drug is to be decidedly preferred to the bromids in those cases in which it can be used advantageously, because no toxic symptoms follow its free administration and the mental faculties are not impaired by its use. [Hebetude is quite marked even after moderate doses. Except in epilepsy, its effects are by no means comparable to those of the bromids.]

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[1] Arch. Internat. de Pharm. et de Therap., vol. IX, fasc. 5 and 6, 1902.

[2] Deut. med. Woch., April 17, 1902.

[3] Am. Jour. Med. Sci., Dec., 1901.

[4] Ophthal. Rec., Oct., 1901.

[5] Phila. Med. Jour., May 3, 1902.

Spartein.—Thomas[1] has experimented very largely with this drug in chronic affections of the heart. He finds that it is a reliable diuretic although it does not, as a rule, stimulate to the secretion of more than 3 liters of urine daily. It is thus inferior to theobromin, although frequently more potent than digitalis. In its action upon the heart it closely resembles digitalis, although it is a much weaker drug. It does not act rapidly. Its cumulative and toxic effects are slight. The principal indications for its use are chronic myocarditis, commencing asystole, subjective cardiac disturbances, and arrhythmia. For advanced cases, and for cases complicated by extensive hepatic and pulmonary changes, the more powerful drug, digitalis, is preferable. The dose should not exceed $\frac{1}{3}$ of a grain in 24 hours by mouth, or $\frac{1}{10}$ of a grain hypodermatically, twice repeated. [More serious investigation is needed for this remedy. Laboratory workers usually assign it a place near the saponins, while clinicians give it a far greater therapeutic value.]

Strophanthus.—Martinet[2] sums up the action of this drug as follows: It cannot be compared to digitalis in the constancy of its effects. Nevertheless, in certain cases it succeeds when digitalis has failed, and it is of value in replacing digitalis during the periods of treatment in which that drug is suspended for the purpose of avoiding its cumulative effect. It has no marked action upon either the vessels or the arterial pressure. It is, therefore, preferable to digitalis in all cases in which arterial tension is evidently a source of embarrassment to an enfeebled heart. The dosage of strophanthus is very irregular. In order to avoid its toxic effects, and at the same time secure its real benefits, it should be given in progressively increasing doses, till the physiologic indication has been fulfilled. There are only two preparations of value—namely, the extract and strophanthin; the tincture is very unreliable. [The variability of the tincture doubtless explains some of the failures ascribed to the remedy.]

Sucramin.—Bellier[3] finds that sucramin presents the following characteristics: It is very soluble in water and slightly soluble in alcohol. Its sweetening power is 700 times greater than that of beet-sugar. Sucramin presents all the characteristics of saccharin, except solubility in the solvents of this substance. By boiling the aqueous solution of sucramin with magnesia considerable ammonia is formed, which shows that it is simply the ammoniacal salt of benzoic sulfamid or saccharin.

Sulfur.—Richmond[4] states that sulfur, both from its solidity and nonabsorbability, is an ideal intestinal antiseptic. When administered in **dysentery,** the stools become less offensive, and flatulence markedly decreases. It is his custom to administer sulfur with opium in acute dysentery, and in chronic cases to use sulfur alone. The manner of the action of sulfur in the intestines is a matter of some doubt. It may be stated that sulfureted hydrogen and other sulfur acids are formed and prohibit the growth of the specific microorganisms. Stengel[1] reports 2 cases of amebic dysentery of long duration, in which sulfur (10 grains 3 or 4 times daily) with opium gave excellent results.

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[1] Rev. Méd. de la Suisse Rom., Mar., 1902.

[2] Ann. Policlin. de Paris, Jan., 1902.

[3] Bull. Gén. de Thérap., Oct. 23, 1901.

[4] Lancet, Nov. 23, 1901.

Suprarenal Extract.—[See also Adrenalin.] Bates[2] repeats his former view that suprarenal extract is a safe, certain, and powerful astringent, hemostatic, and heart stimulant; that its effect is always temporary, and while it does not cure any form of disease itself, it is of great advantage as an adjuvant to other remedies.

Floersheim[3] claims that suprarenal extract is indicated in all forms of **organic heart disease** when the action is weak and irregular, and the pulse slow or rapid and of low tension. It is also indicated, according to this author, in cardiac failure from chloroform anesthesia and from overwork, and in many functional disturbances of the circulation.

Longworth[4] has also found suprarenal extract, even when administered hypodermatically, of value as a heart stimulant. [Collapse has followed the hypodermatic use of solutions of suprarenal extract.]

De Schweinitz[5] finds suprarenal preparations of value in **conjunctival hyperemia** due to local irritation, in **acute conjunctivitis** in **blepharospasms and Fruehjahr's catarrh,** in **trachomatous pannus,** and in various types of keratitis and scleritis. They are also of value in promoting the absorption of cocain, eserin, atropin, and pilocarpin, in facilitating the introduction of lacrimal sounds, and in modifying and clearing up corneal opacities following contusions. He finds, however, that they occasionally produce marked irritation with subsequent hyperemia, so that he would advise the use of freshly prepared solutions of the dried gland as being more efficient than adrenalin. If the latter is used, he thinks it should be in very weak solution.

Kyle[6] states that in his own experience about 70 % of the cases of **throat and nose disease** in which he has employed suprarenal gland have been benefited, while in the others the drug has either been worthless or has produced irritating effects or brought about a condition worse than the original one.

Clarke[7] has found the internal administration of suprarenal extract ineffective in **hay-fever.** Concerning its value as a local remedy in this affection, he concludes: In simple vasomotor rhinitis, with no discoverable local abnormality and no general dyscrasia, suprarenal extract gives favorable results in a large proportion of cases. In cases of hay-fever with some local abnormality in the nose, the drug does not act favorably until the abnormal condition is remedied, and then it may be found to be unnecessary. In cases in which there is a rheumatic or allied dyscrasia suprarenal extract does not act so favorably as in uncomplicated cases.

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[1] Proc. Phila. Co. Med. Soc., Sept., 1902.

[2] N. Y. State Jour. of Med., July, 1902.

[3] N. Y. State Jour. of Med., July, 1902.

[4] Brit. Med. Jour., July 19, 1902.

[5] Therap. Gaz., July 15, 1902.

[6] Therap. Gaz., July 15, 1902.

[7] Boston M. and S. Jour., June 19, 1902.

Floersheim[1] claims to have obtained prompt results from the internal administration (5 grains) of suprarenal extract in **hemorrhage.** Forty-five cases are cited, 21 of hemoptysis, 23 of uterine hemorrhage, and 1 of hematemesis.

Gray[2] reports 6 cases of **pneumonia** in which he employed suprarenal extract. In some of these cases he believes that he obtained a reaction suggestive of the abortion of pneumonia. The powerful vasomotor stimulation is cited as an essential element in the action. Three grains were given every 3 hours.

Meara[3] reports 2 cases of very intense **pruritus of the vulva** in which applications of a strong solution of the suprarenal gland afforded prompt and lasting relief.

Tachiol.—Fluorid of silver has recently been introduced under the name of tachiol as an antiseptic agent. According to Durante,[4] of Rome, a solution of tachiol, 1 : 150,000, kills the most resistant pyogenic organisms in one minute. A solution of 1 : 1000 killed anthrax spores, which had resisted steam for 15 minutes, in from 20 to 30 minutes. For disinfecting suppurating cavities and tuberculous joints solutions of 1 : 1000 to 1 : 100 are recommended, and for injecting into the bladder, 1 : 10,000 to 1 : 5000. Tachiol, according to Durante, is unirritating, is much less toxic than corrosive sublimate, and is not affected by albuminous matters.

Tannoform.—Grumme[5] reports very good results from the use of this drug in hyperhidrosis of the feet occurring in military practice. He employed the drug in pure form, dusting the powder freely into the stockings, which were put on after washing the feet and worn for 24 hours. The good effects continued for 3 or 4 weeks.

Thiocol.—This is the potassium salt of guaiacol-sulfonic acid. It contains about 60 % of guaiacol. Its advantages over other preparations of guaiacol and creasote are its freedom from odor and unpleasant taste and its ready solubility in water. Winternitz[6] reports 16 cases of **tuberculosis** in which he used thiocol—8 grains, 3 or 4 times a day—with gratifying results. Maro,[7] Stekel,[8] von Boltenstern,[9] and Vogt[10] also speak favorably of thiocol in the treatment of phthisis.

Eberson[11] reports 11 cases of **pneumonia** successfully treated with thiocol. He gave to children of 10 years, 1 dram in the 24 hours, and to adults, 75 grains. [This dose seems larger than necessary.]

Thiosinamin.—This is a compound produced by acting on the volatile oil of mustard with ammonia. It occurs in colorless, soluble crystals, of a bitterish taste and a garlicky odor. Hebra, Unna, Juliusberg, Crocker and Pernet, and others have recommended it for its reputed action in softening scar tissue in keloid, postlupus scarring, cicatricial strictures, and deformities. It may be given hypodermatically every other day, in doses of from 1 to 2 grains, dissolved in glycerin and water, or by the mouth in daily doses of 3 grains. Another method of application is by the local use of thiosinamin plasters (10 % to 30 %). Hebra has warned against the use of the drug in all cases of partially healed tuberculous foci. Teleky[1] reports several cases of **cicatricial stenosis of the esophagus** treated with thiosinamin. The results were surprisingly satisfactory in some cases, although not in others, and study of the details indicates that the drug has a distinct softening effect upon old cicatricial tissue. He also cites a case in which a healed gastric fistula became loosened and the stomach separated from the abdominal wall under the influence of thiosinamin, which was being administered for stenosis of the esophagus, with fatal result. Upson[2] has employed thiosinamin in 6 cases of **chronic joint affections.** The results are not conclusive. In 2 cases there was marked improvement; in 2 cases the effect was doubtful, and in 2 entirely negative. Beck,[3] from his experience in 14 cases, finds that injections of thiosinamin do not prove very effective in **chronic ear diseases** unless aided by mechanical treatment, but that when electrolysis and thiosinamin injections are used conjointly the eustachian bougie can be passed with much greater ease and inflation is much easier than without these agents. Suker[4] sums up the results of his experience with thiosinamin in **affections of the eye.** Exudative choroiditis, 6 cases; improvement in 4, none in 2. Corneal opacities, very dense, 10 cases; improvement in 6, none in 4. Corneal opacities, nebulous, 8 cases; marked improvement in 4, moderate improvement in 2, no improvement in 2. Plastic iritis, 6 cases; benefit in 2, slight benefit in 1, no benefit in 3. Capsular cataract, 3 cases; no improvement in any case. Ectropion (moderate cicatricial), 3 cases; improvement in 2, no improvement in the other. Unna[5] states that in a case of **leprosy,** after marked improvement had been attained with potash inunctions, he administers thiosinamin to aid the reabsorption of the lepromas. After a few injections black and blue spots developed on the face and limbs and all the cicatricial patches softened and ulcerated with deep necrosis. A condition bordering on collapse followed which required the free use of stimulants. The general condition soon improved and all the ulcerations healed rapidly, without keloid tissue, so that there is not a trace of the former extensive tuberous cutaneous lepra.

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[1] Med. News, Jan. 4, 1902.

[2] Med. Rec., April 5, 1902.

[3] Merck's Archives, May, 1902.

[4] Lancet, Feb. 8, 1902.

[5] Deut. militärärztl. Zeit., No. 12, 1901.

[6] Deut. Aerzte-Zeit., Jan. 1, 1902.

[7] Rivist. de med. Cir. Pract., Jan. 20, 1902.

[8] Centralbl. f. d. ges. Therap., XIX, No. 5, 1902.

[9] Reichs. medizinal. Anzeiger, XXVI, No. 12, 1902.

[10] Rev. de Therap., LXVIII, No. 24, from Merck's Archives, April, 1902.

[11] Aerztl. Centr.-Zeitung, No. 8, 1902

Thymus Gland.—Mendel[6] has treated more than 100 **rachitic children** with thymus extract during the last 5 years, each child receiving as many grams of fresh gland substance as he was months old, or 6 to 12 tabloids a day. No ill effects were observed; on the contrary, the author states, the rachitic process was favorably influenced in many cases and the functional disturbances were most rapidly and effectively controlled. The author was prompted to employ this treatment on account of the great similarity between the symptoms exhibited by dogs whose thymus glands have been removed and those observed in rachitis, and also because Friedleben found that the thymus gland was abnormally small or abnormally large and hard in all cases of pronounced rachitis which he examined postmortem.

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[1] Wien. klin. Woch., Feb. 20, 1902.

[2] Amer. Med., Jan. 25, 1902.

[3] Laryngoscope, June, 1902.

[4] Jour. Am. Med. Assoc., Aug. 9, 1902.

[5] Therap. der Gegenwart, July, 1902.

[6] Münch. med. Woch., Jan. 28, 1902.

Tropacocain.—Schwarz[1] reports 100 cases of spinal tropacocainization. In a very few cases after-effects were evident, but so brief and so mild that they were in no way comparable to those liable to follow the injection of cocain. He prefers this method for appropriate cases to any kind of inhalation narcosis. He follows Tuffier's technic, and injects $\frac{5}{6}$ of a grain of tropacocain dissolved in 16 minims of water, then elevates the pelvis for 10 minutes, after which he lowers the patient to the horizontal plane. Analgesia always lasted 1 or 2 hours for the legs, genitalia, perineum, and arms. He does not recommend spinal analgesia for abdominal operations. Neugebauer[2] also regards tropacocain in quantities of $\frac{5}{6}$ to 1 grain as a certain and safe analgesic for the lower extremities, but for more highly situated regions, inconstant and unreliable. He reports 60 cases in detail.

Trunecek's Serum.—Leopold-Levi[3] reports 24 cases of **arteriosclerosis, aneurysm, and chronic rheumatism** in which marked improvement followed the use of Trunecek's serum, which is a solution of sodium sulfate, chlorid, phosphate, and carbonate, with potassium sulfate, in water. This is given in hypodermatic injections of 1 cc. every 3 or 4 days, increasing to 5 to 7 cc. or in rectal injections of 35 cc. In another communication Leopold-Levi[4] states that the serum acts by lowering blood-pressure and in some way changing the composition of the blood. The latter, he holds, is certainly the case in chronic rheumatism in which the injections cause immediate improvement. In still another communication[5] he reports the further history of the patients referred to in the early part of the year, and states that all have continued to improve. He also reports a number of new cases and states that he has been equally successful when he administered the alkaline salts in rectal injections or in a powder by the mouth. He slightly alters the formula for the powder, using a mixture of 10 grams of sodium chlorid; 1 gram of sodium sulfate; 75 grams each of calcium and magnesium phosphate; 40 grams of sodium carbonate and 0.3 gram of sodium phosphate. This makes 13 powders, each containing as much salt as 150 cc. of blood-serum. It is taken in a tablespoonful of water an hour before breakfast, and, if necessary, again before lunch.

Tuberculin.—Trudeau[6] states that he has been using tuberculin both for diagnostic and therapeutic purposes for 10 years and has never seen it exert a bad influence on an existing tuberculous process. He believes that its therapeutic sphere is limited, and that it is chiefly valuable in just those cases which are amenable to and are preferably treated by climatic and hygienic treatment. Wilkinson[1] states that he has used tuberculin in 70 cases of tuberculosis and does not remember a single instance in which there was no improvement, provided there was no mixed infection. Hager[2] also has used tuberculin for 10 years. He states than in 30 cases of pronounced tuberculosis treated with tuberculin the results were so striking that this method of treatment forms one of the brightest pages in his experience. He prefers the new tuberculin.

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[1] Münch. med. Woch., Jan. 28, 1902.

[2] Wien. klin. Woch., xv, Nos. 50–52, 1901.

[3] La Presse Méd., No. 5, 1902.

[4] La Presse Méd., No. 6, 1902.

[5] La Presse Méd., No. 49, 1902.

[6] Therap. Monthly, Mar., 1902.

Tuberculocidin.—Jessen[3] has administered Klebs's tuberculocidin in 36 cases of tuberculosis, and states that essential improvement was evident in 63.9 %. All in the first stage were cured, and 54 % of the 11 in the second, and 36 % of 11 in the third stage. The remedy was not entirely free from by-effects. Higher temperature was observed in a few cases and repeated hemoptysis followed in 2 instances. On the whole, however, it deserves more general adoption, he thinks, as it certainly neutralizes to some extent the toxin and thus gives the organism a chance to defend itself.

Urea.—In a second article [see YEAR-BOOK for 1902] on the use of urea in the treatment of tuberculosis, Harper[4] reports 7 cases treated with urea with remarkable success. The preparation used was a synthetic product. At first 10 to 15 grains are given thrice daily, and then the dose is gradually increased to a maximum of 60 grains thrice daily. No harmful effect was observed in any instance. The treatment is based upon the apparent immunity of gouty subjects against tuberculosis.

Urotropin.—Götzl and Salus[5] contend that the efficiency of urotropin as a urinary antiseptic does not depend upon the liberation of formaldehyd at the body-temperature. In a series of experiments they ascertained that at a temperature of 63° F. a 5 % solution of urotropin will kill the microbe of ammoniacal cystitis, Bacillus coli, and the bacillus of typhoid fever. At 97° F. a 0.5 % solution will delay fermentation in an exposed urine as well as a 3 % solution at 63° F.

Urotropin Quinate.—De la Camp[6] has found this remedy useful in gout, especially when there are uric acid concrements. [Observations, not yet completely tabulated, seem to support this statement.]

X-rays.—Morton[7] gives the following answers to the query "What is accomplished by the x-rays in the treatment of malignant growths?" Relief from excruciating pain and constant suffering, often immediately; reduction in the size of the new-growth; establishment of the process of repair; removal of the odor, if present; cessation of the discharge; softening and disappearance of lymphatic nodes; removal of the cachectic color of the skin; improvement in the general health; cure, up to date, of a certain number of malignant growths. Pusey[8] reports 36 cases, including lupus vulgaris, epithelioma, carcinoma of the breast, deep-seated carcinoma, sarcoma, and keloid. The history of each case is given in detail. The results in superficial lesions (lupus and epithelioma) were uniformly successful. Of 5 cases of cancer of the breast, 2 were apparently treated with success. The results were negative in 6 cases of deep-seated carcinoma of the head and neck. The only result obtained in the treatment of abdominal cancer was seemingly some amelioration of pain. A case of sarcoma of the jaw improved almost to cure; 2 other cases of sarcoma showed no results except relief from pain. Pusey concludes his paper by laying down the following propositions: (1) In all cases of malignant disease which have been operated on there is reason to urge the subsequent use of the x-rays as a prophylactic measure. (2) In all inoperable cases of malignant disease the x-rays should be tried. (3) In all such cases there is a probability of relieving pain and a possibility of inhibiting the progress of the disease. Zeit[1] concludes from a very thorough investigation of the subject that x-rays have no direct bactericidal properties, and that clinical results must be explained by other factors, possibly the production of ozone, hypochlorous acid, extensive necrosis of the deeper layers of the skin, and phagocytosis. Rieder[2] also concludes from his studies that the x-rays have no very decided bactericidal activity in man. Coley[3] concludes that the x-rays have a remarkable inhibitory action upon the growth of all forms of malignant disease, and that this is especially true of sarcoma. Lancashire[4] reviews the effects of x-rays and finds them of special value in hypertrichosis, coccogenic sycosis, lupus, and rodent ulcer. Morris and Dore[5] point out the relative advantages and disadvantages of the x-ray and the Finsen light treatment. The latter is tedious and costly, but they do not consider the x-ray an adequate substitute. In the majority of cases, they contend, the Finsen method is more reliable in lupus and rodent ulcer, and apparently gives more permanent results. The effects, moreover, are more easily controlled and the scars better. Pugh[6] reports 4 cases of rodent ulcer cured by x-rays after 38, 36, 26, and 12 applications respectively. Rinehart[7] reports 4 cases of epithelioma of the skin and 1 of nonparasitic sycosis cured by the x-rays. Stuver[8] reports a case of aggravated uterine cancer with ulceration of the vaginal walls and rectum, very extensive discharge, and great debility, in which x-rays were used, a speculum being inserted and the surrounding parts protected with towels and tin-foil. At the time of writing the patient had gained in weight, the pain was greatly ameliorated, the discharges had decreased one-half, and the odor was scarcely perceptible. Sjogren and Sederholm[9] report very favorably upon the use of the x-rays in lupus vulgaris, chronic eczema, vulvar pruritus, and rodent ulcer. Six cases of lupus erythematosus proved very resistant. Eleven cases of hypertrichosis were greatly benefited, but in a certain number there was rapid recurrence.

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[1] Brit. Med. Jour., June 7, 1902.
[2] Münch. med. Woch., July 22, 1902.
[3] Centralbl f. innere Med., No. 23, 1902.
[4] Lancet, Dec. 7, 1902.
[5] Prager med. Woch., Aug. 1, 1901.
[6] Münch. med. Woch., July 23, 1901.
[7] Med. Rec., Mar. 8, 1902.
[8] Jour. Am. Med. Assoc., April 12, 1902.

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[1] Jour. Am. Med. Assoc., Nov. 30, 1901.
[2] Münch. med. Woch., No. 10, 1902.
[3] Amer. Med., Aug. 16, 1902.
[4] Brit. Med. Jour., May 31, 1902.
[5] Ibid.
[6] Brit. Med. Jour., April 12, 1902.
[7] Phila. Med. Jour., Feb. 1, 1902.
[8] Cincinnati Lancet-Clinic, Aug. 16, 1902.
[9] La Semaine Méd., Jan. 8, 1902.

Socolaff[1] has achieved remarkable results with x-rays in the treatment of tinea tonsurans and favus in children. Bondurant[2] reports a case of facial carcinoma and epithelioma, and also one of lupus, treated with advantage by the x-rays. Hopkins[3] reports a number of cases of malignant disease in which the x-rays proved beneficial, and describes the method of applying the treatment.

Yohimbin.—Löwy[4] reports some further favorable results from the use of yohimbin (a mixture of alkaloids from the bark of the Cameroon tree). He found that after castrating dogs, erections set in just as rapidly as with normal animals, so that the action of the drug is not dependent upon hyperemic testes. Williamson[5] reports 3 cases of **neurasthenic impotence** promptly relieved by injections of yohimbin—$\frac{1}{12}$ to $\frac{1}{6}$ of a grain thrice daily. The only disagreeable effects were slight headache and a feeling of nausea, lasting an hour or two. Eulenberg[6] also reports uniform success with yohimbin in neurasthenic impotence. [Unfavorable reports both as to efficacy and even as to untoward symptoms have appeared.]

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[1] Bolnitschnaia Gaz. Botkina, Jan. 23, 1902, from Phila. Med. Jour., April 19, 1902.

[2] N. Y. Med. Jour., Aug. 2, 1902.

[3] Phila. Med. Jour., April 5, 1902.

[4] Therap. der Gegenwart, July, 1901.

[5] N. Y. Med. Jour., Oct. 12, 1901.

[6] Deut. med. Woch., May 29, 1902.

PHYSIOLOGY.

BY G. N. STEWART, M.D.,

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BLOOD.

Hemolysis.—[There is no slackening of the stream of papers on this subject, which presents so many aspects of common interest to the physiologist, the pathologist, and the clinician.] The action of **snake-venom** on the blood-corpuscles has been investigated by S. Flexner and H. Noguchi.[1] The venoms tested differ in hemolytic power. That of the cobra is most active; then follow the venoms of the water moccasin, copperhead, and rattlesnake in the order named. Dog's blood was most quickly and easily destroyed by all the venoms; then followed the blood of the guinea-pig, pig, and rabbit. Snake-venom causes solution of the leukocytes as well as of the colored corpuscles, but there is evidence that the leukolysin is not identical with the erythrolysin. C. Phisalix[2] finds that the resistance of the colored corpuscles of the dog to viper's venom is less than that of the rabbit's corpuscles. This he explains as due to the presence in rabbit's serum of an excess of "anti-hemolysine," a body which is destroyed by heating to 58° or 60° C. Hemoglobin is rapidly transformed into methemoglobin by the poison of the viper, owing to the action of an oxidizing ferment, which he calls "echidnase."

The list of animal hemolytic agents has been swelled by the addition of **spider poison.** According to H. Sachs,[3] NaCl solutions extract from spiders a substance (called by him "arachnolysin") easily destroyed by heating to 70° or 72° C. and possibly identical with the toxalbumin of the spider described by Kobert, and which acts with extraordinary intensity on the colored corpuscles of certain kinds of blood, especially that of the rat and rabbit, while the corpuscles of other animals, as the guinea-pig, horse, sheep, and dog, are not dissolved. Blood naturally immune,—for instance, dog's blood,—when mixed with the solution and then centrifugalized, does not bind any of the poison, which, however, is bound by the stromata of the sensitive varieties of blood, as is required by Ehrlich's side-chain theory.

S. Peskind[4] has investigated the relations of the stromata of the corpuscles to numerous substances which cause their agglutination and precipitation. Most acids and acid salts do so when added to normal or laked blood in minute traces; and the fact that the same reagents produce similar effects on other cells, both animal and vegetable, goes far to vindicate the claim of the stroma to be regarded as the protoplasm of the colored corpuscle. Peskind has also, in conjunction with G. N. Stewart,[1] confirmed and further illustrates the statement of Ransom,[2] that the **antihemolytic body** in serum which protects the corpuscles against saponin is cholesterin. Hédon[3] extends this conclusion to other glucosids (solanin, cyclamin, and digitalin). But, according to him, even when their hemolytic power has been neutralized by cholesterin, their toxicity for the entire animal remains, either because the glucosid-cholesterin compound is still toxic to such cells as nerve-cells, or because it is dissociated in the body.

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[1] Jour. Exper. Med., vol. VI, p. 277.

[2] Compt. rend. de l'Acad. des Sci., t. CXXXV, p. 257.

[3] Hofmeister's Beiträge, Bd. II, S. 125.

[4] Am. Jour. Physiol., vol. VIII, p. 99; ibid., Feb. 1, 1903.

Other interesting bodies belonging to the group of **vegetable blood-poisons,** ricin [the poison of castor oil seeds] and abrin [the poison of the jequirity bean], have been fully investigated by M. Jacoby[4] and W. Hausmann[5] respectively. Jacoby has shown that by treatment with pepsin-HCl the agglutinative action of ricin on the red corpuscles is greatly diminished (thus confirming the statement of Müller),[6] while its general toxic action is unaffected. This is of course strong evidence that the agglutinative and toxic effects are due to separate poisons, as Cushny[7] suggested in opposition to Kobert and Stillmark.[8] Both actions are hindered by immune serum obtained by immunizing rabbits against ricin according to Ehrlich's method. According to Hausmann, abrin is distinguished from ricin by the fact that its agglutinative power is just as resistant to pepsin-HCl as its general toxic power, if not more resistant.

J. Halban and K. Landsteiner[9] have compared the **hemolytic power of fetal and maternal serum.** The latter is capable of dissolving more blood-corpuscles and of producing greater agglutination of them than the serum of the child, and is also more copiously precipitated by "immune" serum. Carré and Valleé[10] state that the hemolytic power of the serum of the ox, sheep, goat, dog, ass, and horse on the blood-corpuscles of the guinea-pig runs parallel with the general toxic action of the serum on this animal; and when the hemolytic action is inhibited by saturation with blood-corpuscles, the general toxic action is inhibited also. They produce evidence that (in the case of the serum of the ox and sheep)[11] the toxic effect on guinea-pig's corpuscles is due to substances which come from dying leukocytes.

E. Hédon[12] has discovered the interesting fact that although [as is well known] rabbit's serum has scarcely any effect on dog's corpuscles *in vitro*, yet if dog's corpuscles, washed perfectly free from serum, and suspended in isotonic NaCl solution, be transfused into the veins of rabbits whose own corpuscles have been for the most part removed by two copious preliminary bleedings, a strong **hemolytic power for dog's corpuscles** is developed in the rabbit's serum, and the animals die in 4 days. [This may be considered an acceleration of the process by which an "immune " serum is obtained by repeated injections of foreign blood into the peritoneal cavity.]

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[1] Jour. of Med. Research, vol. VIII, p. 268.
[2] YEAR-BOOK, Medicine, 1902, p. 571.
[3] Jour. internat. de Pharm. et Thérap., t. IX, p. 393.
[4] Hofmeister's Beiträge, Bd. I, S. 51.
[5] Ibid., Bd. II, S. 134.
[6] Arch. f. exper. Path. u. Pharm., Bd. XLII, S. 302.
[7] Ibid., Bd. XLI, S. 439.
[8] Arb. d. pharmakol. Instit. zu Dorpat, 3 (1889).
[9] Münch. med. Woch., 1902, No. 12, S. 475.
[10] Compt. rend. Soc. de Biol., t. LIV, p. 176.
[11] Ibid., p. 154.
[12] Arch. de Méd. Exper., t. XIV, p. 297.

A. S. F. Grünbaum[1] states that by the **biologic test**, as usually applied, human blood is practically indistinguishable from the blood of the anthropoid apes (gorilla, orang, chimpanzee), the antiserum of the blood of any one of these animals producing a precipitate in the blood of any other, which is practically the same both quantitatively and qualitatively. If, however, the test be made in the same way as in serum diagnosis, it is possible to differentiate them, since an earlier and more complete reaction is obtained when the antiserum is added to its own blood. He lays stress on the interesting proof which the similarity of the reaction affords of the close "blood " relationship between man and these higher monkeys.

Sachs[2] has shown that the results of M. Matthes[3] on the **laking of corpuscles** fixed by Hayem's solution are to be, largely at any rate, explained by the removal of mercury from the corpuscles by the laking agents. Matthes[4] admits that this is the case as regards some of his experiments, but brings forward others which he does not think susceptible of such an explanation. G. N. Stewart[5] has demonstrated that certain of the laking agents (saponin, sapotoxin, bile-salts) produce a marked increase in the permeability for electrolytes of formaldehyd-fixed colored corpuscles and also of pus-corpuscles. This increase of permeability is due to an action on substances soluble in ether (cholesterin and perhaps lecithin). While this increase of permeability may contribute to the liberation of hemoglobin by overthrowing the balance of osmotic pressure, the primary action of the laking agents is the change produced in the relation between the hemoglobin and the stroma. Some of the substances which cause laking of the red corpuscles also affect the leukocytes. Yet the intensity of the action of any given hemolytic agent need not be the same for the two kinds of cells. For example, sodium taurocholate is a more energetic leukolytic agent (in the case of dog's blood) than sapotoxin, while sapotoxin is a more powerful erythrolytic agent. In Necturus's corpuscles **intraglobular crystallization** is produced with great readiness by laking agents, owing, we may suppose, to the disturbance of that equilibrium on which the state of colloid solution of the hemoglobin in the corpuscles depends. When crystallization occurs in the corpuscles the crystals, in general, take a different form from that which they assume outside of the corpuscles, as if the process of crystallization went on in the interior of the corpuscles under constraint of the colloid medium.

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[1] Lancet, Jan. 18, 1902.

[2] Münch. med. Woch., Feb. 4, 1902.

[3] Ibid., Jan. 7, 1902.

[4] Ibid., April 29, 1902.

[5] Jour. of Med. Research, vol. VIII, p. 268; Jour. Exper. Med., vol. VI, p. 257; Am. Jour. Physiol., vol. VIII, p. 103.

Osmotic Relations of the Blood-corpuscles.—D. Calugareanu[1] states, as was previously shown by G. N. Stewart,[2] that salts can enter or leave the corpuscles independently of the hemoglobin. H. J. Hamburger[3] comes to the conclusion that while the colored corpuscles are impermeable to Na ions [as Gürber and others have shown], they are permeable to the electronegative ions of all Na salts, which are exchanged for electronegative CO_3 set free in the corpuscles by the dissociation of their K_2CO_3. In another paper by Hamburger and H. J. van der Schroeff[4] this result is extended to leukocytes and the cells of lymph-glands. G. Manca and G. Catterina,[5] following up work previously reported by the former[6] on nonnucleated corpuscles, have investigated the changes which take place in the osmotic resistance of nucleated corpuscles (of bird, fish, and reptile) when blood is kept aseptically. While there is little change in the first 24 hours, a great part of the corpuscles lose their hemoglobin in 6 to 8 days.

CO-Hemoglobin.—Frau Ferchland and E. Vahlen[7] assert, on the basis of experiments on frogs, that coal-gas is much more poisonous than pure CO. The common idea that coal-gas poisoning is simply CO poisoning is, according to them, far from the truth. Benedicenti[8] found that in a tunnel on the Turin-Genoa Railway the air might contain CO even to the amount of 0.8 % to 0.9 %, a quantity quite sufficient to cause death if for any reason the trains were delayed longer than usual in the tunnel. As a matter of fact, several cases of asphyxia have occurred within the last few years in the tunnels of this line. Instigated by the practical importance of the subject, G. Hüfner[9] has redetermined the law of partition of hemoglobin between O and CO for the temperature of 37.5° C. He gives a curve from which one can reckon for any given percentage of CO in the air the proportion between hemoglobin combined with CO and hemoglobin combined with O: *e. g.*, when CO is present in the air, only to the extent of 0.05 %, 27 % of the hemoglobin is united with CO.

Coagulation of Blood.—[The output of work on this topic has not been so great as in most recent years.] The most interesting contributions are those of M. Arthus[10] on the detection and estimation of fibrin ferment by fluorid plasma, *i. e.*, the plasma of blood to which sodium fluorid has been added to the amount of 3 parts in 1000. The addition of fibrin ferment to this plasma causes clotting, the amount of fibrin formed in a given quantity of plasma in a given time increasing with the quantity of fibrin ferment added. Blood mixed with the fluorid at the moment of abstraction does not contain fibrin ferment, and the addition of the fluorid at any time after the blood has been drawn arrests at once the formation of the ferment. Fibrin ferment, absent entirely from the circulating blood, is rather slowly produced in normally shed blood till near the moment of coagulation, when the rate of its formation is notably augmented. Nor does the production of fibrin ferment stop with this event. On the contrary, it still goes on for several hours, a much greater quantity being formed after than before coagulation. C. Fleig and M. Lefébure[1] have shown that the addition of more than a certain amount of a Ca salt to blood hinders instead of favoring coagulation [an observation which the author of this abstract can confirm].

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[1] Compt. rend. Soc. de Biol. t. LV, p. 460; Centralbl. f. Physiol., Bd. XVI, S. 240.

[2] Jour. of Physiol., vol. XXIV, p. 211.

[3] Arch. f. (Anat. u.) Physiol., 1902, S. 492.

[4] Ibid., Supp., 1902, S. 119.

[5] Arch. di Farmacol. sper., I, Fasc. 2, 1902.

[6] Arch. ital. di clin. Med., 1896.

[7] Arch. f. exper. Path. u. Pharm., Bd. XLVIII, S. 106.

[8] La Respirazione nelle Gallerie, etc., by A. Mosso.

[9] Arch. f. exper. Path. u. Pharm., Bd. XLVIII, S. 87.

[10] Jour. de Physiol. et Path. gén., t. III, p. 887; t. IV, p. 1.

Blood-formation.—J. Gaule[2] asserts that in balloon ascents the number of blood-corpuscles, as enumerated by the Thoma-Zeiss apparatus, is increased, while the hemoglobin is diminished. [Such observations would be of little value as indicating absolute changes in the blood, were it not that Gaule positively states that the character of the corpuscles was altered, numerous nucleated forms being seen in microscopic preparations. This result, so far as it goes, corroborates the conclusion of Jaquet,[3] that there occurs at high altitudes an absolute increase in the total number of corpuscles as well as in the number per cubic millimeter.] H. J. A. van Voornveld,[4] from numerous observations on human beings at Davos Platz, and a critical study of the very voluminous literature, is constrained to pronounce in favor of Jaquet's contention. It is, however, a curious commentary on the difficulty either of making or of interpreting such experiments, that J. E. Alderhalden,[5] an investigator well skilled in blood-analysis, reiterates his previous assertion[6] that the alterations in the number of the corpuscles and the hemoglobin content are relative and not absolute. He could discover no changes in the microscopic character of the corpuscles which could be supposed to support the view that increased formation of them had taken place. [In spite of this, however, it would seem that the greatest weight of evidence is now in favor of an absolute increase.]

H. Stassano and F. Billon[7] confirm Danilewsky's observation that the **erythrocytes are increased in number** after intravenous injection of lecithin. Their resistance to hypoisotonic salt solutions is also increased, and the same is true of the white corpuscles, especially the mononuclear leukocytes.[7] [These facts are interesting in connection with the undoubted rôle which cholesterin, and in all probability lecithin also, plays in the hémolytic action of many substances.] On the other hand, J. Vogel has found that in chronic poisoning of fowls by small doses of phosphorus a marked diminution takes place in the absolute number of the red corpuscles, accompanied by an increase in the excretion of bile.

LYMPH.

F. A. Bainbridge[8] states that **intravenous injection of Na-taurocholate** or of pure hemoglobin causes an **increased formation of**

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[1] Ibid., vol. IV, p. 615.

[2] Pflüger's Archiv, Bd. LXXXIX, S. 119.

[3] YEAR-BOOK, Medicine, 1902, p. 572.

[4] Pflüger's Archiv, Bd. XCII, S. 1.

[5] Zeit. f. Biol., Bd. XLIII, S. 443.

[6] Ibid., Bd. XLII, S. 125.

[7] Compt. rend. Soc. de Biol., t. LIV, pp. 156, 167; Compt. rend. de l'Acad., t. CXXXIV, pp. 318, 430

[8] Jour. Physiol., vol. XXVIII, p. 204.

lymph in the liver, an observation which he holds a confirmation of the theory of lymph-production put forward by Asher.[1] [Asher[2] obtained a similar result on injection of ox-bile. And it is questionable whether the introduction into the blood of Na taurocholate, a laking agent which causes not only erythrolysis but marked leukolysis, is really a "simpler" experiment, as Bainbridge supposes. It certainly does not obviate completely his objection to Asher's procedure, that bile contains nucleoproteid, since the decomposition of the blood-corpuscles must liberate this substance.] The statement of Asher and Barbéra, repeated in a recent paper by Asher,[3] that peptone [a well-known lymphagog], when intravenously injected, has a marked cholagogic action, is denied by Bainbridge.[4] The explanation of this contradiction is given by A. Ellinger,[5] who finds that peptone only increases the outflow of bile from the gall-bladder and not its secretion. Leech extract, another lymphagog, has no cholagog action. [It is, therefore, difficult to see how Asher can make good his contention that in all cases the formation of lymph is due to the physiologic activity of the organs.]

L. B. Mendel and D. R. Hooker[6] confirm the observation of Clopatt[7] that **extracts of the strawberry have a lymphagogic action,** not due to the crystalloid substances contained in them. They have noted a postmortem flow of lymph after intravenous injection of such extracts. In one animal this lasted for 4 hours after death. They point out [very justly] that this indicates that the processes on which the formation of lymph depends are not purely physical, but are related to the physiologic activity of cells, in this case of surviving cells.

HEART AND CIRCULATION.

Vasodilators.—W. M. Bayliss[8] makes the remarkable announcement that the only nerve-fibers acting as vasodilators for the hind-limb (and intestine) are posterior root-fibers, which conduct the impulses antidromically, *i. e.*, away from their cell-bodies. These fibers can be excited reflexly; *e. g.*, by stimulation of the depressor.

The **blood-flow in the external jugular vein** has been measured by the aid of Hürthle's recording stromuhr[9] by R. Burton-Opitz,[10] who obtained in a 13-kilo dog a mean velocity of 147 mm. per second. The volume of the blood-stream was nearly trebled by section of both vagi.

Residual Blood-pressure after Arrest of the Circulation.—L. Hill[11] failed to find evidence that a mean hydrostatic pressure of the blood (determined by the capacity of the vascular system and the quantity of blood in it) actually exists, as has been assumed by various writers. On arresting the circulation, the residual aortic pressure is neither the same, nor due to the same causes, as the residual pressure in the venæ cavæ. According to MacWilliam,[1] the condition of the vascular system after death by cardiac failure varies widely according to the means used to paralyze the heart: (1) The condition usually associated with asphyxia may be present, *i. e.*, engorgement of the right side of the heart and the systemic veins, while the left side of the heart and the arteries are relatively empty; or (2) both ventricles may be contracted and practically empty; or (3) there may be much blood in both sides of the heart. The characteristic asphyxial condition of the heart, as seen some hours after death, is a postmortem phenomenon, in the production of which the early onset and relatively great intensity of rigor mortis in the left ventricle plays an important rôle. According to H. C. Wood, Jr.,[2] the rise of pressure produced by asphyxia in the pulmonary artery is caused by contraction of the pulmonary vessels due to stimulation of the vasoconstrictors of the lesser circulation.

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[1] YEAR-BOOK, 1899, p. 952.

[2] Zeit. f. Biol., 1899, S. 261.

[3] Centralbl. f. Physiol., Bd. XVI, S. 203.

[4] Loc. cit.

[5] Hofmeister's Beiträge, Bd. II, S. 297.

[6] Am. Jour. Physiol., vol. VII, p. 380.

[7] Skand. Arch. f. Physiol., Bd. X, S. 403.

[8] Jour. Physiol., XXVIII, p. 276.

[9] Proc. Internat. Cong. of Physiol., Turin, 1901.

[10] Am. Jour. Physiol., vol. VII, p. 435.

[11] Jour. Physiol., vol. XXVIII, p. 122.

The Cerebral Circulation.—H. Cushing[3] believes he has obtained evidence that the vasomotor center regulates the arterial pressure in correspondence with the intracranial pressure, so that an increase in the latter occasions such an increase in the arterial pressure that it tends to find a level slightly above that of the pressure exerted against the medulla. Owing to this mechanism, a degree of cerebral compression corresponding to an intracranial pressure much above the normal blood-pressure can occur without causing symptoms.

The Heart.—D. J. Lingle[4] brings forward some new facts in support of Loeb's idea that NaCl is of fundamental importance for the rhythmic activity of the heart. He shows that this salt is absolutely necessary for the origination of rhythmic contractions in isolated strips cut from the turtle's ventricle. He combats Howell's statement[5] that NaCl without the presence of Ca cannot start the beats, and asserts that the reason why in NaCl solutions the beats come to an end is probably the lack of O. In this connection the observation of Magnus.[6] that the activity of the excised mammalian heart can be maintained for a considerable time when gaseous O instead of blood is circulated through the coronary vessels, acquires additional interest. A portion of the beneficial effect of the oxygen in this case seems to be that it facilitates the removal of gaseous waste products, such as CO_2, since H gas also maintains the beat for a considerable time, while CO_2 stops it at once. The experiments of S. S. Maxwell and J. C. Hill[7] have further accentuated the importance of taking account of the dissolved oxygen in investigating the action of solutions on living tissues. Howell's argument[8] that Ca must be considered indispensable for the automatic contractions of the heart, since they are stopped by sodium oxalate, is replied to by Anne Moore,[1] who states that sodium sulfate, which equally with sodium oxalate precipitates Ca, restores the beat of the frog's lymph hearts and the rhythmic contractions of toad's skeletal muscles after they have been exhausted in NaCl.

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[1] Ibid., vol. xxvii, p. 337; Brit. Med. Jour., April 5, 1902.

[2] Am. Jour. Physiol., vol. vi, p. 283.

[3] Arch. Ital. de Biol., 1901; Mitt. aus d. Grenzgeb. d. Med. u. Chir., Bd. ix., S. 773; Johns Hopkins Hosp. Bull., Sept., 1901.

[4] Am. Jour. Physiol., vol. viii, p. 75.

[5] Ibid., vol. vi, p. 181.

[6] Arch. f. exper. Path. u. Pharm., Bd. xlvii, S. 200.

[7] Am. Jour. Physiol., vol. vii, p. 409.

[8] Ibid., vol. iv, p. 195.

A. Kuliabko[2] has demonstrated anew the **extraordinary tenacity of life of the cardiac muscle** by restoring to pulsation by the artificial circulation of Locke's fluid[3] (which contains only inorganic constituents of the blood, with 0.1 % of dextrose) a rabbit's heart which had been kept 44 hours in the ice-chest in complete standstill. In further experiments he obtained pulsations, at least in certain parts of the heart, after an interval of 3 to 5 days in animals; while 20 hours after death from double pneumonia the heart of a boy, 3 months old, was restored to pulsation, and went on beating for over an hour.

Heart-nerves.—T. W. Engelmann[4] develops further his idea that in the cardiac nerves three sets of fibers exist: inotropic fibers, which influence the amount of mechanical work performed by the heart; dromotropic fibers, which influence its power of conducting the contraction; and bathmotropic fibers, which affect the excitability of the cardiac muscle, as measured by the intensity of the weakest stimulus which is capable of causing contraction. [In spite of the eminence of the author of this theory, and the very numerous and often lengthy papers in which he has defended it, we agree with H. E. Hering[5] that no real proof has been given of the independent existence of three such properties of the cardiac muscle, much less of the independent existence of three such sets of nerve-fibers.]

RESPIRATION.

Von Stejskal[6] finds support for v. Basch's views[7] on the bad effect on respiration of congestion of the lungs due to cardiac lesions in his experiments, which show clearly that when the quantity of blood in the lungs is increased their extensibility diminishes, and with this diminution in extensibility the proportion between the respiratory pressure and the amount of air taken in becomes smaller.

R. Magnus[8] asserts that the **lungs are practically impermeable for ammonia.** As he points out, this is of interest as showing that the pulmonary membrane does not permit gases to diffuse through it indifferently, but selects them [a result which agrees with the recent work of Bohr, Haldane and Smith, and others].

A. Desgrez and V. Balthazard[9] have adapted to the respiration of human beings the method of regenerating air by sodium peroxid previously described by them.[1] The oxygen formed replaces that used up in respiration, while the NaOH formed absorbs the CO_2. They recommend the adoption of this method for the ventilation of sick-rooms and the cabins of ships, and in diving operations.

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[1] Ibid., vol. vii, p. 315.

[2] Pflüger's Archiv, Bd. xc, S. 461; Centralbl. f. Physiol., Bd. xvi, S. 330.

[3] Locke: Jour. Physiol., vol. xviii, p. 332; Centralbl. f. Physiol., Bd. xv, S. 600. Göthlin: Skand. Arch. f. Physiol., Bd. xii, S. 1. Bergström: Centralbl. f. Physiol., Bd. xvi, S. 202. Schücking: Ibid., Bd. xv, S. 681

[4] Arch. f. (Anat. u.) Physiol., 1902, S. 103, 443; Ibid., Supp. Bd., 1902, S. 1.

[5] Pflüger's Archiv, Bd. xcii, S. 391.

[6] Ibid., S. 327.

[7] Cf. Year-Book, Medicine, 1902, p. 574.

[8] Arch. f. exper. Path. u. Pharm., Bd. xlviii, S. 100.

[9] Jour. de Physiol. et Path. gèn., t. iv, p. 497.

DIGESTION.

The most remarkable recent work on the secretion and properties of the digestive juices is that of Pawlow[2] and his pupils. Synopses of this work or of portions of it have been published by Carvallo,[3] C. J. Martin,[4] F. A. R. Jung,[5] A. W. Perry,[6] etc. Establishing fistulas of various kinds by a series of most original operations, Pawlow has studied in detail all the digestive secretions, and has revolutionized the current doctrine on many points. Among the most interesting of his results is the proof of the great importance of the psychic condition induced by the sight and taste of appetizing food as an excitant to gastric secretion, an observation which throws a new light on the treatment of dyspepsia and anorexia. Food given by the mouth produces a far more copious flow of gastric juice and is far more rapidly digested than the same food when introduced into the stomach by a gastric fistula. The amount and digestive power of the gastric juice also vary to some extent with the kind of food when it is taken by the mouth. The passage of the chyme from the stomach into the duodenum is regulated by the reaction of the duodenal contents. So long as this is acid, the pylorus remains closed. As soon as the first small portion of acid chyme ejected from the stomach is neutralized, the pylorus opens again. The neutralization of the chyme in the duodenum is brought about by the increased secretion of the pancreatic juice and the outpouring of bile from the gall-bladder in response to the stimulus of the HCl of the gastric contents. The great function of the bile, according to Pawlow, is to serve as an intermediary to facilitate the passage of gastric into intestinal digestion. It counteracts the action of pepsin, which is dangerous for the pancreatic ferments, and favors the action of those ferments, in particular that of the fat-splitting ferment.

Various investigators have attempted to determine the mechanism by which acid in the duodenum excites the pancreatic secretion. The assumption which lies nearest at hand is that it is a reflex nervous mechanism. And Popielski, one of Pawlow's pupils, whose work in favor of this view was mentioned in a previous report,[7] has brought forward further arguments in its support.[8] Nevertheless, according to Bayliss and Starling,[9] this conclusion seems to be erroneous. They have shown that the contact of acid with the epithelial cells of the duodenum causes

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[1] Ibid., Mar., 1899.

[2] Le travail des glandes digestives, Paris, 1901.

[3] Art. "Estomac," in Richet's Dict. de Physiol., 1902.

[4] Intercol. Med. Jour. of Australasia, May 20, 1902.

[5] Jour. Am. Med. Assoc., May 10, 1902; Med. News, Nov. 9, 1901.

[6] Pacific Med. Jour., Mar., 1902.

[7] Year-Book, Medicine, 1902, p. 579.

[8] Centralbl. f. Physiol., Bd. xv, S. 682.

[9] Proc. Roy. Soc., vol. lxix, p. 352; Jour. Physiol., vol. xxviii, p. 325

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One of the most valuable features of these atlases is that they offer a ready and satisfactory substitute for clinical observation. Such observation, of course, is available only to the residents in large medical centers; and even then the requisite variety is seen only after long years of routine hospital work. To those unable to attend important clinics these books will be absolutely indispensable, as presenting in a complete and convenient form the most accurate reproductions of clinical work, interpreted by the most competent of clinical teachers.

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[3] Ibid., p. 648.
[6] Compt. rend. Soc. de Biol., t. LIV, p. 513.
[7] Zeit. f. physiol. Chem., Bd. XXXIII, S. 451; Bd. XXXV, S. 134; Bd. XXXVI, S. 13.
[8] Ibid., Bd. XXXV, S. 419.

bid., p. 474.
Centralbl. f. Physiol., Bd. XVI, S. 121.
Loc. cit.
Ibid., Bd. XXXV, S. 432.

juice of the dog, and Hamburger and Hekma[1] have found it in the intestinal juice of man. N. Sieber and E. Schumoff-Simonowski[2] have demonstrated that erepsin destroys the diphtheria toxin, which is also destroyed by trypsin.

The **extraordinarily exact adaptation of the digestive juices to the nature of the food** has been one of the most surprising of Pawlow's results. In dogs fed exclusively on meat, the trypsin is present in the pancreatic juice almost entirely in the form of actual ferment and hardly at all as zymogen. In this case the intestinal juice does not increase the proteolytic activity of the pancreatic juice. Indeed it often diminishes it somewhat. On the other hand, in dogs fed essentially with starch and fat (bread, milk, etc.) the pancreatic juice has only a very feeble immediate action on albumin, but is very energetically reinforced by the intestinal juice. Clearly the pancreatic juice in this case contains the trypsin in the form of zymogen. The necessity of such an adaptation Pawlow finds in the fact that active trypsin readily destroys the amylolytic and lipolytic ferments, whose activity must not be interfered with when the diet consists largely of starch and fat. In the intestine, where trypsin is rendered active by enterokinase, the fat-splitting and amylolytic ferments are protected by the proteids of the food and by the bile. The reinforcing action of the bile upon the fat-splitting ferment of the pancreatic juice is similarly shown to depend upon the nature of the food. Pawlow has proved that dog's bile has a slight digestive action on fibrin, and A. Tschermak[3] has extended this observation to human bile. But the main function of bile is to aid the pancreatic juice. Its favorable influence on the pancreatic digestion of albumin, which has already been pointed out by Rachford, has again been emphasized by C. Delezenne.[4]

The **mutual relations of the spleen and pancreas** have formed the subject of numerous inquiries. A. Herzen and P. Pilpoul[5] find it necessary to admit, in the light of Pawlow's discovery of enterokinase, which causes an extrapancreatic transformation of zymogen into trypsin, that in animals deprived of the spleen the intestinal digestion of proteid is not suppressed, but is carried on to an extent sufficient for the needs of the organism. They still, however, uphold the Schiff-Herzen hypothesis, that the spleen produces a substance which plays a part in the elaboration of trypsin within the pancreatic cells. L. F. Rettger[6] and L. B. Mendel and Rettger[7] bring forward some fresh evidence in support of the view that the spleen is concerned in pancreatic digestion; viz., that extracts of the organ prepared from it when congested during digestion exert a favorable influence on the proteolytic power of the pancreas. They are, however, unable to grant to the spleen such an important rôle in this regard as has been claimed for it by Herzen. [The spleen, no doubt, like other organs, contains an enzyme which can aid in the digestion of proteid within its cells,] and J. B. Leathes[1] has shown that the products of the action in an acid medium of the proteolytic enzyme found in the cell-juice from the spleen of the ox are the same as those formed by trypsin in an alkaline medium. [But this is a different thing from affirming that the spleen is specially related to pancreatic digestion.]

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[1] Koninkl. Akad. van Wetensch. Amsterdam, 1902, p. 733; Jour. de Physiol. et Path. gèn., t. IV, p. 805.

[2] Zeitschr. f. physiol. Chem., Bd. XXXVI, S. 244.

[3] Centralbl. f. Physiol., Bd. XVI, S. 329.

[4] Compt. rend. Soc. de Biol., t. LIV, p. 592.

[5] Jour. de Physiol. et Path. gèn., t. IV, p. 625.

[6] Proc. Am. Physiol. Soc., Am. Jour. Physiol., vol. VI, p. xiv.

[7] Am. Jour. Physiol., vol. VII, p. 387.

Bile.—An elaborate investigation of the composition and properties of human bile from 9 cases of fistula has been made by J. Brand.[2] He concludes that fistula bile is always different from normal liver bile, the only condition under which fistula bile could have the same composition as normal bile being that in which as great an amount of bile-acid is introduced into the intestine as escapes by the fistula. The color of normal bile is golden yellow. [This is the ordinary statement, and must, in our opinion, be accepted as final, in spite of the assertion of some authors that human bile is green. The statement that bile has a higher electric conductivity than blood is no doubt correct. But this is not merely because of the high concentration of salts in bile, as the author seems to suppose, but also because of the presence in blood of the nonconducting corpuscles.]

Hay's Test for Bile-acids.—The sulfur test was erroneously described in the last report[3] as Haycraft's instead of Hay's test.

Fat-splitting Ferment in Blood.—Hanriot's[4] statement that the blood contains a lipase, or fat-splitting ferment, has been controverted by M. Doyon and A. Morel,[5] who, continuing a research previously announced,[6] find that sterile dog's blood leaves the alkalinity of a mixture of fat and soda solution unchanged. Hanriot[7] replies that a whole series of fats dissolved in blood are split up. Doyon and Morel[8] admit that when blood is kept aseptically in a thermostat the ethereal extract of the blood does diminish in amount, but find no proof that this is due to fat-splitting. M. Arthus[9] clears up the discrepancy by the discovery that blood-serum contains a ferment (monobutyrinase) which decomposes monobutyrin into butyric acid and glycerin but does not split up ordinary neutral fats. He points out[10] [with justice] that since Hanriot's first experiments were not conducted aseptically, they could prove nothing as regards the presence of a ferment with the power of splitting up neutral fats.

Reaction of Intestinal Contents.--The well-worn question of the reaction of the contents of the intestine has again been taken up by I. Munk,[11] who, after sharply criticizing the methods employed by F. Marquardsen and M. Matthes,[12] arrives at the result that neither in carnivora nor in omnivora, under any conditions of feeding, are the contents of the duodenum, jejunum, or ileum ever alkaline, at least when one uses indicators (such as phenolphthalein) which are sensitive to CO_2 and also to free fatty acids. [It has already been pointed out by the author of this abstract[1] that at least in dogs, 20 to 24 hours after a meal, the contents of all parts of the intestine are acid to phenolphthalein as well as to other indicators.]

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[1] Jour. Physiol., vol. XXVIII, p. 360.

[2] Pflüger's Archiv, Bd. XC, S. 491.

[3] YEAR-BOOK, Medicine, 1902, p. 584.

[4] YEAR-BOOK, 1899, p. 1030.

[5] Compt. rend. Soc. de Biol., t. LIV, p. 614.

[6] Centralbl. f. Physiol., Bd. XV, S. 176.

[7] Compt. rend. Soc. de Biol., t. LIV, p. 655.

[8] Jour. de Physiol. et Path. gèn., t. IV, p. 656.

[9] Ibid., pp. 56, 455.

[10] Ibid., p. 66.

[11] Centralbl. f. Physiol., Bd. XVI, S. 33, 146.

[12] Ver. d. Cong. f. inn. Med., Wiesbaden, 1898, Bd. XVI, S. 258. Cf. Matthes, Centralbl. f. Physiol., Bd. XVI, S. 145.

Movements of Alimentary Canal.—G. Fasola,[2] continuing a research begun by him in conjunction with L. Sabbatani, pronounces absolutely against the existence of antiperistalsis in the intestine. In the light, however, of W. B. Cannon's results, obtained under practically physiologic conditions, this conclusion must be considered too sweeping. Cannon[3] has extended to the study of the intestinal movements in the cat the method previously used by Roux and Balthazard and by himself[4] for the study of the movements of the stomach (mixing subnitrate of bismuth with the food and investigating the movements by the Röntgen rays), and has obtained even more interesting results. "The activity most commonly seen in the small intestine is the simultaneous division of the food in a coil into small segments, and a rhythmic repetition (in the cat at the rate of 30 times a minute) of the segmentation, each time applied to the new segments formed from portions of those just divided. . . . Peristalsis is usually combined with segmentation. . . . The usual movement of the transverse and ascending colon is an antiperistalsis. This recurs in periods about every 15 minutes, and each period lasts commonly about 5 minutes. This antiperistalsis gives new significance to the ileocecal valve, for the food, now in a closed sac, is thoroughly churned and mixed by the constrictions running toward the cecum, and again exposed to absorbing walls without any interference with the processes in the small intestine." No evidence has been obtained of antiperistalsis in the small intestine, but "since the ileocecal valve will allow nutrient material under pressure to pass backward, the antiperistalsis of the large intestine may force into the small intestine a considerable portion of a large enema." The movements continue during sleep. They may be stopped both in the small and large intestine by emotion.

W. P. May[5] has reinvestigated the **action of the vagus and splanchnic nerves on the gastric movements.** For the splanchnics he was unable to demonstrate either an inhibitory or a motor influence. For the vagus his results were very definite. While this nerve exerts no tonic influence on the stomach, stimulation of its peripheral end causes first inhibition and then, after an interval, an increase of tone, and an augmentation of the contractions. Moritz[6] has supplied additional illustrations of the fact that the time of sojourn of the food in the stomach depends on its consistence.

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[1] Manual of Physiology, 4th edit., 1900, p. 353; YEAR-BOOK, Medicine, 1901, p. 550.

[2] Lo Sperimentale, LVI, p. 33; Centralbl. f. Physiol., Bd. XVI, S. 318.

[3] Am. Jour. Physiol., vol. VII, p. 251.

[4] YEAR-BOOK, 1899, p. 957.

[5] Brit. Med Jour., Sept. 13, 1902.

[6] Zeitschr. f. Biol., Bd. XLII, S. 565.

ABSORPTION.

Absorption of Fat.—E. Pflüger[1] has continued the experiments on this subject previously reported.[2] He sums up his conclusions in the statement that in the present state of our knowledge it appears that the fats must be split up into glycerin and fatty acids before absorption. The fatty acids are absorbed in the form of loose compounds; *e. g.*, with the amid group of the bile-acids. In these loose combinations soluble in water the fatty acids can be absorbed from the acid intestinal contents. He lays great stress on the important part played by the bile in the solution of fatty acids.[3] O. Löwi[4] puts forward the hypothesis that fats are absorbed neither as emulsions nor in solution in the contents of the intestine, but that they are dissolved by the lecithin and cholesterin of the epithelial cells. He bases this view on the assertion that Ca soaps, which are quite insoluble in the intestinal liquid, are absorbed from the intestine, while a fat emulsion cannot be observed in the basal membrane of the intestinal epithelium. Pflüger[5] animadverts severely on these experiments and this conclusion, and restates his position in the words, "All digestion is hydrolysis, all absorption is hydrodiffusion." It is doubtful whether this position is shaken by the statement of H. Friedenthal[6] that the colloidal silicic acid is absorbed from the intestine, as well as metallic Hg rubbed up with fat.[7] [Certainly the conclusion that since metallic Hg dissolved in ether and oil, in which it is slightly soluble, can be demonstrated by no reagent for Hg, and that none of the Hg can therefore be ionised, while it is in accordance with the current view that all instantaneous chemical reactions are reactions of ions, cannot be accepted without reserve in the light of the observation of L. Kahlenberg[8] that instantaneous chemical reactions—*e. g.*, the precipitation of cupric chlorid by HCl gas from a solution of cupric oleate in pure benzene—are possible in nonconducting solutions as well as in solutions of electrolytes.]

Absorption of Sugar.—[While the demonstration that the fats are absorbed in solution is a satisfactory step toward a physico-chemical explanation of absorption, the general question of the nature of the absorptive powers of the intestinal mucous membrane remains shrouded in darkness.] E. W. Reid,[9] *e. g.*, summing up the result of careful experiments on the absorption of glucose, says: "It appears from a study of the absorption of weak solutions of glucose that with the intestinal membrane as normal as the experimental procedure will permit, phenomena present themselves which are as distinctly opposed to a simple physical explanation as those previously studied in the absorption of serum.[10] Probably with weak solutions of nonirritating substances the specific cell-action is the main factor." J. Nagano,[1] too, finds that the velocity of absorption of various simple sugars is different. This is the case even for stereoisomeric sugars, *i. e.*, sugars whose molecule, with the same number of atoms combined in the same way, has a different form. Reach[2] states that sugar is absorbed from rectal enemas in smaller amount than has been supposed, and dextrin to a still smaller extent than sugar, while starch is only slightly and slowly absorbed.

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[1] Pflüger's Archiv, Bd. LXXXVIII, S. 431.

[2] YEAR-BOOK, Medicine, 1902, p. 581.

[3] Pflüger's Archiv, Bd. LXIX, S. 211.

[4] Sitz. d. Ges. d. Naturwiss., Marburg, June, 1901.

[5] Pflüger's Archiv, Bd. LXXXIX, S. 211.

[6] Arch. f. (Anat. u.) Physiol., 1902, S. 149.

[7] Pflüger's Archiv, Bd. LXXXVII, S. 467.

[8] Zeitschr. f. physikal. Chem., Bd. VI, S. 1; Nature, Nov. 13, 1902.

[9] Jour. Physiol., vol. XXVIII, p. 241.

[10] YEAR-BOOK, Medicine, 1902, p. 581.

Absorption of Proteids.—O. Cohnheim[3] discusses the supposed regeneration of peptone to serum- or organ-proteids in the intestinal wall in the light of his discovery of the peptone-splitting ferment, erepsin, and is led to the conclusion that the disappearance of peptone in contact with the mucous membrane of the intestine, first observed by Salvioli and then by Hofmeister, is not due to its regeneration, but to its decomposition into the well-known simple products yielded also by action of trypsin and acids. [It is obvious that this is a factor which has henceforth to be reckoned with in making deductions from such experiments], and the announcement of Cohnheim[4] that in octopods (Octopus vulgaris and Eledone moschata) proteid is absorbed in the intestine in the form of crystalline products (leucin, tyrosin, lysin, arginin) and NH_3 is of interest in this connection. While he is careful not to extend this result without special investigation to vertebrates, he surmises [and rightly so] that from the great similarity of the conditions it is probably true for them also. [The decomposition of proteids preliminary to absorption seems accordingly to be much more thorough and far-reaching than has been hitherto supposed.]

URINE.

[The mechanism of urinary secretion is one of those problems which never seem to lose their fascination for the physiologic observer. A large amount of work has again been devoted during the past year to this subject, and especially to what may be considered the simplest division of it, the secretion of the inorganic constituents.] A. R. Cushny[5] brings forward certain facts which he believes can be most naturally explained on Ludwig's theory, that a dilute urine is filtered through the glomeruli, and that this urine is altered by the absorption of water (and other substances) as it passes along the urinary tubules. For instance, it is well known that when NaCl is withheld from an animal, the NaCl in the urine is greatly diminished. This diminution may be accounted for on the supposition that the glomeruli cease to secrete NaCl when the proportion of it in the blood sinks below a certain amount, or on the supposition that while still filtered off from the blood by the glomeruli, it is reabsorbed by the renal epithelium. Now, Cushny finds that the intravenous injection of sodium sulfate causes an increase in the percentage of NaCl in the urine of a rabbit whose tissues are poor in NaCl. He explains this as due to the smaller opportunity for reabsorption of NaCl in the tubules when the flow of urine along them is accelerated by the diuresis that follows the injection of the sulfate. When NaCl and Na_2SO_4 are simultaneously injected into the blood, the two salts do not influence each other's excretion, the same amount of each being excreted as if it had been injected by itself, a fact also pointed out by W. Ruschaupt.[1] W. Filehne[2] criticizes Cushny's reasoning, and concludes that his results may be reconciled with the vital theory as well as with the mechanical. Ruschaupt,[3] too, has found that during diuresis caused by one salt in a rabbit enriched with another salt, an absolutely greater quantity of the latter salt is excreted. And W. Ercklentz[4] has observed an influence of injected NaCl on the excretion of sodium chlorate previously introduced. Further, T. Sollmann,[5] as previously reported, saw that after the injection of isotonic solutions of Na_2SO_4 the Cl almost disappeared from the urine. [So that it would be rash to conclude that Cushny's observations have placed Ludwig's theory on a firm basis.] B. Haake and K. Spiro[6] also produce evidence that the dissolved substances in the blood exert specific diuretic actions on the renal mechanism, apart from their osmotic pressure. They slowly injected into the veins small quantities of various solutions isotonic with the blood-plasma. Of all the substances injected, NaCl caused the smallest diuresis. And, in general, a substance not normally present in the blood produced more diuresis than a normal constituent. In a rabbit rendered poor in NaCl the injection of an isotonic solution of NaCl produced no diuresis for a considerable time, but, on the contrary, a diminished flow of urine, while a similar solution injected into the veins of a rabbit previously fed with salted food caused an immediate and considerable diuresis. T. Sollmann and E. D. Brown,[7] in an extensive research on the excretion of egg-albumen after intravenous injections, observed, too, that the albumin, apart altogether from the effects of the solvent, exerts a specific diuretic action.

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[1] Pflüger's Archiv, Bd. xc, S. 389.

[2] Arch. f. exper. Path. u. Pharm., Bd. xlvii, S. 231.

[3] Zeitschr. f. physiol. Chem., Bd. xxxiii, S. 396. [4] Ibid., Bd. xxxv, S. 396.

[5] Proc. Am. Physiol. Soc., Am. Jour. Physiol., vol vi, p. xvii: Jour Physiol., vol. xxvii, p. 429.

A. Gurwitsch[8] assaults the mechanical theory from another side. For instance, after ligation of the renal portal vein of one kidney in male frogs, he finds that the flow of urine from that kidney is much diminished as compared with the other. Since the renal portal vein supplies the tubules, and the circulation through the glomeruli is not affected, he argues that if Ludwig's theory were correct the opposite result ought to be obtained, the excretion of water not being interfered with and its reabsorption being stopped. Repeating and extending the experiments of Nussbaum, he has also injected various pigments into the blood after ligation of one renal portal vein. In the corresponding kidney the lumen of the convoluted tubules contains little or no pigment. He concludes, therefore, that in the intact kidney the pigment in the epithelium must have come not from the lumen of the tubules but from the blood of the renal portal vein. He observes [quite justly] that v. Sobiéranski's statement that after injection of pigment the epithelial cells are colored with the pigment in the neighborhood of the lumen merely shows that these cells, like many others, have a considerable capacity for taking up pigments such as indigo, and not that they absorb water from the lumen.

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[1] Pflüger's Archiv, Bd. xci, S. 574.
[2] Ibid., S. 565.
[3] Loc. cit.
[4] Ibid., S. 599.
[5] Year-Book, Medicine, 1902, p. 583.
[6] Hofmeister's Beiträge, Bd. ii, S. 149.
[7] Jour. exper. Med., vol. vi, p. 207.
[8] Pflüger's Archiv, Bd. xci, S. 71.

W. Lindemann[1] has endeavored to exclude the glomeruli in mammals by injecting oil through the renal artery. After a short time, according to him, the oil emboli clear away from practically all parts of the kidney except the glomeruli, which remain plugged. If indigocarmine be subsequently injected into the blood, it is not only taken up from it by the embolized kidney as well as by a normal one, but is excreted. The quantity of urine is markedly diminished and its specific gravity increased, but its composition is not essentially altered. He concludes that the tubules are in a high degree independent of the glomeruli as an apparatus for the excretion of urine, and looks upon his results as an additional proof of the Bowman-Heidenhain theory.

M. Pfaundler[2] and L. Schwarz[3] have investigated the changes produced in the urine by an increase of pressure in the ureter or by its ligation, with essentially similar results. By obstruction the molecular concentration of the urine is in all cases diminished to ½ to ¾ of the normal. This diminution is only slightly due to a deficiency of urea. An increase of pressure in the ureter leads, in general, to an increased secretion of urine.

Physical Chemistry of Urine.—Numerous papers on the freezing-point and electric conductivity of urine have appeared, among them papers by M. Chanoz and C. Lesieur[4] on the cryoscopic study of the urine in normal individuals; by P. Nobécourt and G. Delamare[5] on the cryoscopy of the urine in pregnant women, normal and albuminuric; and by A. Steyrer[6] on the so-called "osmotic analysis" of urine. The last-named observer admits that this new method does not yield much more information for clinical purposes than ordinary chemical analysis. A good and critical summary of all that is of most interest to the practitioner is given by T. Sollmann.[7]

METABOLISM, NUTRITION, AND DIETETICS.

The Liver and NH_3 Poisoning.—[Nencki, Pawlow, and Zaleski, on the strength of experiments made by means of Eck's fistula (an artificial connection between the portal vein and the inferior vena cava), propounded the doctrine that it is one of the functions of the liver to prevent NH_3-poisoning by transforming the NH_3 compounds of the blood into urea. They found that animals subjected to this operation developed symptoms which they interpreted as that of NH_3-poisoning when fed on a flesh diet; and they demonstrated that, as a matter of fact, the blood of the portal vein normally contains 3 to 5 times as much NH_3 as arterial blood.] And although W. Horodynski, S. Salaskin, and J. Zaleski[8] have recently shown that the method used by Nencki and his co-workers gives absolute values for the NH_3 which are much too high, they find by an improved method that the ratio between the quantity in the portal and that in the arterial blood was correctly stated by the older observers. Nevertheless, it would appear from the work of A. Biedl and H. Winterberg[1] that the assumption of an autointoxication by NH_3 after interference with the passage of the blood through the liver, cannot be accepted. For with artificial injection of NH_3 and NH_4 salts into a vein larger quantities of NH_3 must be present in the blood, in order to produce symptoms, than are present even in the portal blood after the establishment of Eck's fistula; and, further, the quantity of NH_3 which must be present in the portal blood, when injection is made into a branch of the portal vein, in order that the symptoms of NH_3-poisoning may appear is the same as that required in the arterial blood when the NH_3 is introduced into an ordinary vein. [A point which seems to have been overlooked in this investigation is the permeability of the colored corpuscles for NH_4 salts. We know that such salts as NH_4Cl penetrate the corpuscles very freely, and, once within them, are, within certain limits at any rate, withdrawn from active interchange with the plasma. Now, it is to be supposed that it is the NH_3 compounds in the plasma, and not those in the corpuscles, which give rise to toxic symptoms. We cannot, therefore, make any definite statement as to the effective concentration of the NH_3 compounds in the blood, if we know merely the amount of NH_3 contained in the entire blood. And it is quite conceivable that in the case of NH_3 compounds absorbed into the blood from the intestine during digestion of proteids, the effective concentration in the plasma may be greater for a given percentage of NH_3 in the entire blood than for the same percentage when NH_3 salts are artificially injected.

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[1] Zeitschr. f. Biol., Bd. xlii, S. 161.

[2] Hofmeister's Beiträge, Bd. ii, S. 336.

[3] Centralbl. f. Physiol., Bd. xvi, S. 281.

[4] Jour. de Phys. et Path. gèn., t. iv, p. 865.

[5] Ibid., t. iii, p. 993.

[6] Hofmeister's Beiträge, Bd. ii, S. 312.

[7] Amer. Med., Oct. 25, 1902, p. 656.

[8] Zeitschr. f. physiol. Chem., Bd. xxxv, S. 246.

Katherine Kowalesky and S. Salaskin,[2] in continuation of their work on the **formation of uric acid in the liver of birds,**[3] have investigated the NH_3 and lactic acid content of the blood of these animals. They find that the NH_3 content is higher than in dog's blood, and especially constant. It increases under the influence of fatal doses of acids, but is not sensibly altered by large doses of alkalies. The lactic acid is very little affected either by acids or alkalies. As regards the formation of uric acid, they draw the general conclusion that no essential difference exists between birds (and reptiles) and mammals (and all other animals). In all animals the uric acid is partly formed synthetically and partly by oxidation.

Glycolytic Power of Blood.—F. W. Pavy and R. L. Siau[4] have re-investigated the question of the glycolytic power of drawn blood. Working with aseptic precautions, they find that the loss of sugar even in several hours is never large. Their results lend no support to Lépine's theory of diabetes.

Phloridzin Glycosuria.—[It is known that after subcutaneous injection of phloridzin more sugar is excreted in the urine than when the drug is given by the mouth.] O. Loewi[1] has shown that the difference is due to the nonabsorption of a portion of the phloridzin or its active decomposition products in the alimentary canal. The dose of phloridzin which gives the maximal excretion of sugar with a given diet must be increased when the sugar-forming substances in the food are increased. As to the source of the sugar in phloridzin glycosuria, G. Lusk[2] states that none of it is produced from fat. The heat equivalent of the sugar excreted is equal to that of the increased amount of proteid decomposed.

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[1] Pflüger's Archiv, Bd. LXXXVIII, S. 140.

[2] Zeitschr. f. physiol. Chem., Bd. XXXV, S. 552.

[3] Ibid., Bd. XXXIII, S. 210.

[4] Jour. of Physiol., vol. XXVII, p. 451.

A new form has been added by J. Kóssa[3] to the almost interminable list of glycosurias produced by drugs, viz., **chromic acid glycosuria,** induced by the subcutaneous administration of most compounds of chromic acid and especially by potassium chromate. The condition belongs to the group of so-called renal glycosurias, since, as in phloridzin glycosuria, the sugar-content of the blood is not increased when the excretion of the urine is interfered with.

According to Cadeác and Maignon,[4] a transient glycosuria or glycuronuria (appearance of glycuronic acid in the urine) occurs after ligation or crushing of muscles in the guinea-pig, or the tearing of muscles in the fracture of bones in man.

Seat of Formation of Sugar from Proteids.—[While the formation of sugar from proteids is thoroughly established, the seat of the transformation in the body has not been definitely located, although Seegen[5] has suggested that it is the liver.] So far as they go, the observations of F. Kraus[6] lend no support to this idea. He finds that when blood is artificially circulated through a liver rich in glycogen, the sugar of the blood increases. If the liver has been previously rendered poor in glycogen, the increase is very small. And in neither case is the increase of sugar influenced by the addition of Witte's peptone to the blood.

Glycogen Formation.—[It is a question of interest whether in anaerobic animals like the intestinal parasites the metabolism is fundamentally different from that of aerobic animals.] E. Weinland and A. Ritter[7] have put this question to the test in the case of the formation of glycogen, a subject on which a mass of information exists in regard to the higher animals. They conclude that in Ascaris, upon the whole, there is no fundamental difference, glycogen being formed from the same substances as in aerobic animals.

P. Jensen[8] has investigated the **glycogen metabolism of the heart.** In the starving dog the heart contains the same amount of glycogen as in the normal animal, at a time when in the skeletal muscles the proportion has sunk to $\frac{1}{10}$ to $\frac{1}{30}$ of the normal. [But these results need revision in the light of Pflüger's recent work on glycogen estimation. Working with his improved method, Pflüger found a considerable amount of glycogen left in starved animals. Jensen's conclusion that the heart-muscle has the power of maintaining its glycogen store under conditions in which this is not possible for the skeletal muscles, while not improbable in itself, is further weakened by the fact that in the hearts of some of his animals he found no glycogen at all, a result which he seems to pass over without sufficient criticism. He discusses the question whether all the glycogen in the body would suffice to afford the energy needed for the work of the heart, if the cardiac muscle used up only glycogen, and answers it in the affirmative. But apart altogether from the fact that for the purposes of his argument he assumes the total circulation time in a 13 kilo dog to be one minute, which is too long, he makes no allowance for the large amount of energy transformed into heat in the cardiac muscular fibers, an amount certainly greater than that which is represented by the mechanical work.]

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[1] Arch. f. exper. Path. u. Pharm., Bd. XLVII, S. 48.

[2] Zeitschr. f. Biol., Bd. XLII, S. 31.

[3] Pflüger's Archiv, Bd. LXXXVIII, S. 627.

[4] Compt. rend. de l'Acad., t. CXXXIV, p. 1000.

[5] Zuckerbildung im Thierkörper. 1900, S. 246.

[6] Pflüger's Archiv, Bd. XC, S. 630.

[7] Zeitschr. f. Biol., Bd. XLIII, S. 490.

[8] Zeitschr. f. physiol. Chem., Bd. XXXV, S. 514, 525.

Fat Metabolism.—A. S. Loevenhart,[1] starting with the discovery made by himself in conjunction with Kastle[2] that the action of lipase (the fat-splitting ferment of the pancreas) is reversible, which has been confirmed by Hanriot[3] for the synthesis of monobutyrine from butyric acid and glycerin, discusses the relation of lipase to the metabolism of fat in the body. He sees in this action of lipase a clear explanation of fat absorption. Lipase has been shown to occur in all the tissues of the body in which it has been looked for, especially in the liver, the active mammary gland, the blood, the lymph, and the intestinal mucosa. It is of particular interest, in connection with the rôle he attributes to this ferment, that it is present in considerable amount wherever synthesis of fat is known to take place, *e. g.*, in the subcutaneous fat and in active mammary gland. The enzyme is either a fat-splitting or a fat-forming ferment according to conditions.

Oxidizing ferments, or oxidases, the occurrence of which in certain secretions and in the cells of certain organs has now been established, are awakening widespread attention. Among the latest papers on the subject are those of O. Loew[4] on one of these, which he calls catalase, and which has the power of decomposing H_2O_2; and of Raudnitz,[5] who announces the existence of such a ferment in milk. R. Lillie,[6] using a color reaction, first employed by Ehrlich in 1885, as an indicator of oxidations in the body, has obtained microscopic evidence that the nucleus plays a predominant part in intracellular oxidation, possibly by means of oxidizing ferments.

Iron-content of Hen's Egg.—[Various attempts have been made to increase the amount of iron in hen's eggs by feeding with iron preparations.] C. Hartung[7] has compared the iron content of normal eggs with that of eggs produced under administration of citrate of iron. While a certain though not a great increase is produced, this takes place only after a long time. Thus 100 grams of egg-substance contained 4.4 mg. of Fe_2O_3 before the administration of iron was begun; after feeding with iron for 3½ weeks the amount was 4.5 mg., after more than 2 months 7.4 mg., and after a year 7.3 mg. He concludes that this is not a practical commercial method of obtaining eggs rich in iron, and recommends rather the feeding of the fowls with foods naturally rich in iron.

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[1] Am. Jour. Physiol., vol. vi, p. 331.

[2] Am. Chem. Jour., vol. xxiv, p. 491.

[3] Compt. rend. Soc. de Biol., 1901, p. 70.

[4] Rep. No. 68, U. S. Dept. of Agric., May, 1901; Zeit. f. Biol., Bd. xliii, S. 256.

[5] Ibid., xlii, S. 91.

[6] Am. Jour. Physiol., vol. vii, p. 412.

[7] Zeitschr. f. Biol., Bd. xliii, S. 195.

Alcohol.—Kassowitz,[1] after passing in review the evidence accumulated up to the present time, comes to the conclusion that although alcohol is burnt in the body [since Atwater's work[2] nobody can doubt this], it is not a food, and should not be used for the purpose of nutrition either in health or disease. F. S. Lee and W. Salant[3] find that on the isolated skeletal muscle of the frog it exerts in medium doses a favorable, and in large doses an unfavorable, action.

INTERNAL SECRETION.

Suprarenals.—H. Cristiani and A. Cristiani[4] have studied the effects of total and partial extirpation of these organs in rats. Even a very small portion of the capsules is sufficient to permit the animals to live, if it includes a certain amount of medullary substance in good condition. Even a relatively large amount of cortex does not prevent death if medullary substance be absent. V. Schmieden[5] announces, in a preliminary communication, that he has solved the hitherto baffling problem of successfully grafting portions of an extirpated suprarenal in other organs of the body of the same animal (rabbit).

Suprarenal Diabetes.—F. Blum[6] has followed up his discovery that in the suprarenal there is a substance which, when it gets into the circulation, causes glycosuria.[7] Very small quantities of suprarenal cause glycosuria when administered subcutaneously, as do also suprarenin and adrenalin. Herter[8] had previously found large quantities of sugar (even over 9 %) in the urine after adrenalin. Zuelzer[9] has confirmed these results, and Metzger[10] has shown that the sugar-content of the blood is markedly increased, in the rabbit up to more than 1 %, and in the dog up to about 0.7 %. This increase is produced by interference especially with the glycogen store in the liver, and the glycosuria rarely occurs in starved dogs. It therefore resembles the puncture glycosuria rather than pancreatic or phloridzin diabetes. A. C. Croftan[11] has also observed the phenomenon, and has shown that the suprarenal contains a diastatic ferment[12] in large amount, which, he suggests, may be connected with the suprarenal glycosuria, and possibly with the pigmentary changes that so frequently accompany disease of the suprarenal capsules, since he has lately discovered evidence[13] that the formation of normal bile-pigment (and bile-acids) from hemoglobin through the action of the "liver ferment" can take place only in presence of dextrose or glycogen.

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[1] Pflüger's Archiv, Bd. xc, S. 421.
[2] YEAR-BOOK, Medicine, 1901, p. 555.
[3] Am. Jour. Physiol., vol. VI, p. xiii; Ibid., vol. VIII, p. 61.
[4] Jour. de Phys. et Path. gèn., t. IV, p. 837.
[5] Pflüger's Archiv, Bd. xc, S. 113.
[6] Ibid., S. 617.
[7] Deut. Arch. f. klin. Med., Bd. LXXI, Oct., 1901.
[8] N. Y. Med. Jour., 1902.
[9] Berl. klin. Woch., No. 48, 1901.
[10] Münch. med. Woch., No. 12, 1902.
[11] Amer. Med., Jan. 18, 1902.
[12] Pflüger's Archiv, Bd. xc, S. 285.
[13] Phila. Med. Jour., Jan. 11 and Jan. 18, 1902.

Nervous and Other Tissues.—S. Vincent and W. Sheen[1] confirm the statement of Osborne and Vincent[2] that extracts of nervous tissue when injected intravenously cause a fall of blood-pressure, not due to cholin, as supposed by Halliburton,[3] but to an active substance of unknown nature which can be extracted by alcohol, though not by ether. Extracts of muscular tissue also produce a distinct temporary fall of blood-pressure, though not so great as in the case of extracts of nervous tissue. Saline decoctions of other tissues (testis, kidney, spleen, pancreas, liver, mucous membrane of stomach and intestine, lung, and mammary gland) all cause a fall of blood-pressure. The medulla of the suprarenal and the infundibular portion of the pituitary body appear to be the only tissues which yield a pressor substance. To these must be added, on the authority of A. Bied and J. Wiesel,[4] the so-called accessory organs (*Nebenorgane*) of the sympathetic, two bodies lying in the retroperitoneal space, one on each side of the aorta, as described by E. Zuckerkandl.[5] Extracts of these bodies have essentially the same action as suprarenal extracts.

ANIMAL HEAT.

F. G. Benedict and J. F. Snell[6] have published an elaborate and valuable series of **temperature observations on human beings** under various conditions. They used electric resistance thermometers of the kind described by them in a previous paper.[7] [Their complaint that Pembrey[8] has, without sufficient examination, discredited the practical usefulness of such thermometers seems justified. They have been employed with success, among others, by Masje[9] and G. N. Stewart,[10] although, curiously enough, Benedict and Snell describe these observers as having used the thermoelectric method.] Among other interesting results of this research are the absence of any appreciable tendency to reversal of the daily curve of temperature when work was done in the night and rest taken in the day, and the rapid rise of temperature produced by muscular work. The latter has been noticed also in an extensive series of observations on normal monkeys by S. Simpson,[11] who saw that a chase of half an hour might raise the rectal temperature by as much as 3° to 4° F. W. O. Atwater[12] has continued his work on the statistics of metabolism and heat-production by means of his respiration calorimeter. His latest observations are well summarized by N. Zuntz.[13] He extends Rubner's demonstration of the conservation of energy in the animal body to the most varied forms of nutrition and to the condition of hard muscular work.

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[1] Jour. Physiol., vol. XXVIII, p. xix.

[2] Ibid., vol. XXV, p. 283.

[3] Cf. YEAR-BOOK, Medicine, 1902, p. 591.

[4] Pflüger's Archiv, Bd. XCI, S. 434.

[5] Ver. d. anat. Ges., Bonn, May, 1901.

[6] Pflüger's Archiv, Bd. XC, S. 33.

[7] Ibid., Bd. LXXXVIII, S. 492.

[8] Schäfer's Textbook of Physiology, I, p. 830.

[9] Virchow's Archiv, Bd. CVII, pp. 17, 267.

[10] Studies from the Physiol. Lab., Owen's College, Manchester, 1891, p. 100.

[11] Proc. Physiol. Soc., Jour. Physiol., vol. XXVIII, p. xxi.

[12] Bull. 69 and 109, U. S. Dept. of Agric.

[13] Biochemisches Centralbl., Bd. I, S. 1, Dec. 15, 1902.

MUSCLE, NERVE AND ELECTROPHYSIOLOGY.

Among the numerous important papers which have appeared, mainly from Loeb's laboratory, on the **relations of ions to life processes,** the following may be mentioned: Anne Moore[1] has investigated the effect of solutions of electrolytes and nonconductors on rigor mortis and heat rigor, and shows that although the entrance of water favors coagulation, and lowers the coagulation temperature, the coagulation phenomena cannot be explained entirely on the basis of osmosis. The nature of the ions present is a determining factor. She finds support for the conclusion of v. Fürth and Stewart and Sollmann that only two proteids capable of coagulation have been clearly demonstrated in striped muscle.

H. Cushing,[2] in addition to other observations on the poisonous effect of pure NaCl solutions on the muscle-nerve preparation, has confirmed the result of F. S. Locke[3] that the abolition by pure NaCl solutions of the indirect excitability of the muscle-nerve preparation is due to a paralyzing action on the motor nerve-endings, an action which can be counteracted by solutions containing Ca salts.

W. D. Zoethout[4] has further investigated the phenomenon discovered by Loeb and named by him **contact irritability** (viz., that a frog's muscle previously treated with a Na salt whose anion precipitates Ca is excited by contact with such substances as oil, water, air, etc., unlike a normal muscle). He concludes that, as Loeb suggested, contact irritability depends on the disturbance of the normal ratio between the salts in the muscle, possibly the ratio between the Ca and K salts.

R. S. Lillie[5] has studied the action of various inorganic salts and mixtures of such on ciliary and muscular movement in the larvae of certain marine annelids (Arenicola and Polygordius), and announces that each of the chlorids, NaCl, $MgCl_2$, and KCl, exerts a specific influence on the contractile tissues, from which he infers that each salt forms with the tissues a dissociable salt-proteid compound, an ion-proteid in Loeb's sense.[6] K salts form compounds much less dissociable than the others, a fact which may explain their peculiarly poisonous effects.

Overton[7] has made an extensive study of the **permeability of striped muscular fibers** to numerous substances. He finds that so long as the fibers are uninjured they are permeable or impermeable for exactly the same compounds as other animal and vegetable cells. All substances easily soluble in mediums like ether or olive oil readily penetrate the fibers. In a second paper[8] he reports that Na (or in a minor degree Li) ions are indispensable for the contraction of skeletal muscle. [He very curiously omits all reference to the work of Loeb and his pupils, which has thrown so much light on the relation of these and other ions to muscular contraction in general. His general argument, that the living muscular fibers must be, at any rate in the resting condition, relatively impermeable for the salts of the blood-plasma, on the one hand, and those of the fibers themselves, on the other, since these salts are qualitatively different throughout life, has been previously stated and extended to all cells by the author of this abstract,[1] and adopted by J. S. Macdonald[2] in his discussion of the nature of the injury current of mammalian nerve.[3]]

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[1] Am. Jour. Physiol., vol. VII, p. 1.

[2] Ibid., vol. VI, p. 77.

[3] Centralbl. f. Physiol., Bd. VIII, S. 166; Ibid., Bd. XV, S. 573.

[4] Am. Jour. Physiol., vol. VII, p. 320.

[5] Am. Jour. Physiol., vol. VII, p. 25.

[6] Ibid., vol. III, p. 327.

[7] Pflüger's Archiv, Bd. XCII, S. 115.

[8] Ibid., S. 346.

J. Loeb[4] has shown that the toxic effect of a pure solution of NaCl, LiCl, NH_4Cl, or KCl on the **development of Fundulus embryos** and (in conjunction with H. Neilson[5]) on striped muscle can be abolished by the addition of small quantities of certain salts with a bivalent cation (Ca, Sr, Ba or Mg salts) or with a trivalent cation (Al or Cr salts). He believes that the toxic and antitoxic effects are functions of the electric charge of the ions, but comes to the opposite conclusion as regards the stimulating and inhibitory action of ions on muscles.[6] M. Bial[7] states that the inhibitory power exerted by acids on fermentation by yeast depends on the content of H ions, while the anions of the acids are without significance.

The Nerve Impulse.—H. Boruttau[8] has continued his discussion of the theory of the conduction of the nerve impulse and the relation of the action current to the core-conductor structure of nerve. A. P. Mathews[9] has attempted to explain the nerve impulse as a propagated change of physical state, a precipitation or jellying of the colloid solutions of the axis-cylinder, produced by a change in their electric condition.

CENTRAL NERVOUS SYSTEM.

Motor Areas.—A. S. F. Grünbaum and C. S. Sherrington[10] have published the results of stimulation and ablation experiments on the motor areas in the higher apes (chimpanzee, orang, and gorilla). These experiments entail important modifications of the current topography of this region. In particular it is worthy of note that no movement whatever was obtained from the ascending parietal convolution, the motor area being bounded posteriorly by the bottom of the sulcus corresponding to the rolandic fissure. The areas are arranged from below upward in an exactly inverse order to that of the corresponding parts of the body: viz., (1) face and head, (2) neck, (3) shoulder, (4) arm, (5) upper part of trunk, (6) lower part of trunk, (7) lower limb. The last-named area, which was small, was confined to the sagittal surface of the ascending frontal convolution.

P. Flechsig[11] has studied the **time of myelinization of the human**

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[1] Jour. Physiol., vol. xxiv, 1899, p. 211; Jour. Exper. Med., vol. vi, p. 259.

[2] Thompson Yates Lab. Rep., Liverpool, 1902, p. 268.

[3] See also Macdonald: Proc. Roy. Soc., vol. lxvii, pp. 310, 315, 325.

[4] Pflüger's Archiv, Bd. lxxxviii, S. 68; Am. Jour. Physiol., vol. vi, p. 411.

[5] Ibid., vol. vii, p. 405.

[6] Pflüger's Archiv, Bd. xci, S. 248.

[7] Zeit. f. phys. Chem., Bd. xl, S. 513; Arch. f. (Anat. u.) Physiol., 1902, S. 361.

[8] Pflüger's Archiv, Bd. xc, S. 233.

[9] Science, Mar. 28, 1902; Century Magazine, 1902.

[10] Proc. Roy. Soc., vol. lxix, p. 206; Brit. Med. Jour., Oct. 12, 1901.

[11] Proc. Internat. Cong. of Physiol., Turin, 1901; Brit. Med. Jour., Oct. 12, 1901; Lancet, Oct. 19, 1901.

cortex, with the result that he is able to distinguish on it no less than 36 areas, every one of which possesses a special anatomic position and therefore a special functional importance. S. I. Franz[1] has investigated in cats the relations of the so-called **association areas of the cerebrum,** and especially the frontal area, to certain acquired habits involving simple mental processes. After a bilateral lesion in the frontal lobes, newly formed but not long-standing habits are lost. This cannot be due to "shock," since other brain lesions are not followed by loss of the habits. Unilateral lesions of the frontal areas usually cause a partial loss of newly acquired habits, or rather a slowing of the association process. The lost habits may be regained, and even again lost, after a second operation, and once more regained.

Sleep.—K. Brodmann[2] has studied the cerebral circulation during sleep in a patient with a defect in the skull over the occipital lobes. He has obtained no evidence that the brain is anemic during sleep. On the contrary, it contains more blood during quiet sleep than in the waking condition. W. Deutsch,[3] after a critical examination of the literature, comes to a similar conclusion.[4]

Decerebrate Rigidity.—A. Fröhlich and C. S. Sherrington[5] have attempted to localize the path which the inhibitory impulses follow in the cord when the triceps muscle, contracted in consequence of removal of the cerebrum, is made to relax. They find a small, fairly limited area in the cross-section of the ventrolateral column, stimulation of which causes relaxation of this muscle combined with contraction of other muscles. The effect is more marked on the side of the stimulation than on the opposite side.

Nerves of Taste.—Sir W. R. Gowers[6] describes 5 cases in which the gasserian ganglion and the adjacent portion of the fifth nerve were removed for neuralgia. In all the loss of taste was complete on that side of the tongue. In explanation of the contrary results obtained by others he says that no weight must be attached to observations made during the first 2 or perhaps 3 weeks after operation, since it is a curious fact that sensation (both taste and common sensation) may persist for some time in the region supplied by the nerve. He sees in these observations complete confirmation of his previous surmise[7] that all the fibers of taste reach the brain by the roots of the fifth nerve. On the other hand, H Cushing,[8] having observed the results of extirpation of the gasserian ganglion in 14 cases, is equally positive that taste is not lost. [It is clear that some factor not hitherto sufficiently studied must be involved when competent observers reach opposite conclusions on a matter of this kind.]

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[1] Am. Jour. Physiol., vol. VIII, p. 1.

[2] Jour. f. Psychol. u. Neurol., Bd. I, S. 10; Centralbl. f. Physiol., Bd. XVI, S. 310.

[3] Wien. med. Woch., 1901, Nos. 32 and 33.

[4] Cf. YEAR-BOOK, Medicine, 1902, p. 594.

[5] Jour. of Physiol., vol. XXVIII, p. 14.

[6] Ibid., p. 300.

[7] Ibid., vol. III, p. 229.

[8] Proc. Am. Physiol. Soc., Am. Jour. Physiol., vol. VIII, p. 27.

REPRODUCTION.

F. P. Gorham and R. W. Tower[1] assert that the **action of KCN in prolonging the life of the eggs of the sea-urchin,** observed by J. Loeb and W. H. Lewis,[2] is due to its antiseptic influence on the sea-water and not to a specific favorable effect on the eggs. They believe that the only demonstrated action of KCN on the eggs is a poisonous one. E. P. Lyon[3] shows that the resistance of the eggs to KCN diminishes as they develop, similar changes being brought about by lack of O. Numerous additional papers of great interest on artificial parthenogenesis have been published by Loeb and his pupils; *e. g.*, by A. W. Greeley,[4] M. H. Fischer,[5] A. P. Mathews[6] and J. Loeb, M. Fischer, and H. Neilson,[7] but the limits of our space permit us only to mention them.

[1] Am. Jour. Physiol., vol. VIII, p. 175.

[2] Ibid., vol. VI, p. 305.

[3] Ibid., vol. VII, p. 56.

[4] Ibid., vol. VI, p. 296.

[5] Ibid., vol. VII, p. 301.

[6] Ibid., vol. VI, p. 216

[7] Pflüger's Archiv, Bd. LXXXVII, S. 594

LEGAL MEDICINE.

By JOHN MARSHALL, M.D., Nat.Sc.D., and J. H. W. RHEIN, M.D.,

OF PHILADELPHIA.

Epitome.—In forensic medicine there has been very little progress made in the last year. Some interesting contributions have been added to the subject of the recognition of blood-stains by the serum-test, although these have been mainly along the lines proposed and elaborated by Bordet, Nuttall, Uhlenhuth, Wassermann, Schütze, and others, whose work was reviewed in detail in the Year-Book of 1901. The relation that x-ray photographs hold to legal medicine, contributions to the questions surrounding the subject of medical education, examinations for license to practice medicine, and the reciprocity of licensure, are the most important matters considered during the past year in this department of medicine.

LEGAL DISCUSSIONS UPON MEDICAL QUESTIONS.

An important decision was rendered by the Circuit Court of the United States for the Western District of Missouri[1] in the case of the **Knights Templars' and Masons' Life Indemnity Company vs. Rosa Jarman.** The policy had been issued to John P. Jarman, husband of the respondent, by the above-named company in October, 1885, and provided in case of self-destruction of the holder of the policy, voluntarily or involuntarily, sane or insane, that the holder of the policy would then have no hold upon the company. It also provided that in case of the suicide of the policy-holder the heirs were entitled to an amount on his policy equal to the amount paid to the company in assessments, without interest. The amount specified by the policy to be paid in event of death was $5000 plus all the money paid in assessments and subject to certain restrictions, which were as follows: "Provided that a policy of membership for $5000 shall be good for all the money in the death-fund, arising from one assessment, providing it does not exceed $5000, and all the money paid on the policy in assessments; and that a certificate for $4000 shall be good for four-fifths of all the money in the death-fund arising from one assessment, providing it does not exceed $4000, and all the money paid in assessments, and so on, in proportion as to all certificates." In January, 1889, and again in January, 1894, the company amended the section just quoted, so that upon the death of a member of the company his heirs should receive only the amount set forth in the policy, that is to say, the assessments should not be returned. The case was tried upon an agreed statement of facts. The petitioner claimed that the respondent was entitled to recover only the amount of assessments paid by the insured, who had been guilty of self-destruction. The second defense was that the respondent was entitled to recover only $5000 by reason of the amendment in the constitution in 1894. The respondent contended that the Indemnity Company at the time that the policy was issued was a regular life insurance company, subject therefore to certain statutes of Missouri which provided that the **suicide clause** would hold good only when the insured contemplated suicide at the time of applying for the policy. [In the nature of things this part of the statute must be ineffective in the great majority of cases, as it is very unusual for one insuring his life with the idea of defrauding the insurance company to give information of his contemplated suicide prior to the commission of the act.] The respondent further claimed that Jarman did not commit suicide in a technical or popular sense, but died from disease. In reply to the second defense the respondent claimed that the company had no right to reduce the amount of the policy by an amendment of its constitution or by-laws, and that it did not appear that this amendment of the constitution had any retrospective effect, or had more than a prospective operation, as it still provided "that the settlement with the beneficiaries should be made upon the basis of the policy." The petitioner sought to evade the force of the suicide statute by claiming that the act of the Legislature of Missouri of 1887 exempted life insurance companies conducted upon the assessment plan from the insurance laws of the State, and therefore, as far as this contract was concerned, the suicide statute was repealed. It also claimed that "committed suicide" referred to felonious suicide, and therefore the defense of suicide was not within the meaning of the statute, and was therefore not applicable. It was further contended that although the Legislature of 1897 repealed the assessment law of 1887, such law "was void because constitutional requirements as to its method of enactment were not complied with." Judgment was rendered in favor of respondent, for $6013.30 and costs, and this judgment was affirmed by a majority of the judges of the Circuit Court of Appeals, and confirmed by the United States Supreme Court.

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[1] Baltimore Underwriter, Nov. 5, 1902.

MEDICAL JURISPRUDENCE, ETC.

Richardson[1] reviews the **recent Christian Science trial,** and discusses the question of **Eddyism.** In this recent trial in Toronto, he states that it was shown that there was no protection from the law by pleading their peculiar religious beliefs. Mrs. Eddy, in 8 years, graduated 4000 practitioners after a few weeks' course, who were given diplomas. Richardson states that the demonstrations upon which Mrs. Eddy bases her religious beliefs are as follows: (1) An immense number of cases in which there is very little amiss, such as slight accidents; (2) severe injuries which are represented as being fractures or dislocations; (3) hysteric manifestations; (4) diseases of a functional nature; (5) paralytic cases in which the power in the paralyzed part has been restored by nature; (6) cases diagnosed as cancer by those entirely ignorant of every form of disease. Mrs. Eddy claims that she has received her revelation from God, and said her book was the child called Wonderful, which was spoken of by the Prophet Isaiah. Richardson demonstrates clearly that the spread of Eddyism is due to (1) the way in which it disposes of evil; (2) to the fascination attached to anything mysterious or unexplainable; (3) to the lack of care used by so many people in reading a book requiring special study. The language of Mrs. Eddy's book is confusing, and, as Dean Hart says, has the effect of producing a strange maze which dazes the mind; "the reader becomes mentally dizzy—mesmerized, in fact." The writer believes that Christian Science is nothing less than unadulterated quackery.

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[1] Canadian Jour. of M. and S., Dec., 1901, p. 403.

Millican[1] reviews the **Patrick case,** and discusses at some length the expert testimony together with the physiologic and pathologic action of chloroform inhalation.

Lambert Ott[2] discusses the **medicolegal aspect of injuries real and feigned,** and points out how the difference may be detected. He believes that medical testimony should be acted upon by a jury of medical men, who should likewise fix the amount of the damages.

The Appellate Court of Illinois, Third District, in the case of the Chicago and Alton Co. vs. Davis,[3] rules that if a **doctor is employed by a representative of the company** to treat an injury to an employee **the company is liable for the bill.** [Probably suffering is prolonged and possibly deaths occur through the disinclination of employees or representatives of corporations to endeavor immediately to secure the services of a physician in cases of accident to fellow-employees because of fear that, should the physician render a bill for services to the corporation, the displeasure of the corporation will be visited upon them.]

Johnson[4] discusses the question of **medical expert evidence,** and proposes that in order to provide against the danger attending the evidence given by dishonest medical experts, the court should appoint a **medical board of witnesses,** the members of which would be subject to cross-examination just as expert witnesses are under our present practice. [This is an ideal method, and to a certain extent prevails in Germany, but is hardly practicable in this country, where the citizen holds to his right to select his own experts.]

The Court of Appeals of New York[5] holds that the **obligation** on the part of the insured is **only to submit to examination by the medical examiner** and to answer the questions asked by him. If the examiner fails to record the answers as given to him, omitting important points, the responsibility lies with the insurance company, and the beneficiary may show the answers as actually given in order to defeat a forfeiture claimed by the company, on alleging that the answers given were false. The examiner is the agent of the company and not of the insured, although the latter may agree that the physician was his agent and that it cannot be held that he became so in spite of the law and the facts.

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[1] N. Y. Med. Jour., April 5, 1902, p. 587.
[2] Jour. Am. Med. Assoc., p. 295, Nov. 1, 1901.
[3] Jour. Am. Med. Assoc., Oct. 12, 1901.
[4] Canad. Pract. and Rev., Aug., 1901, p. 432.
[5] Jour. Am. Med. Assoc., 1902, p. 895.

The Supreme Court of Michigan[1] is of the opinion that a hospital which is a **charitable institution,** or the trustees thereof, are **not liable for negligence** on the part of the house-surgeon in performing an operation. At the trial it was proved that the house-surgeon was competent in every respect, and that he had received his position by reason of passing a competitive examination, held by a competent medical board. The court further held that, even although the hospital had not been a charitable institution, it had fulfilled every obligation in employing the surgeon. [The ruling that a hospital is a charitable institution and, therefore, is not liable for damages for alleged malpractice has also been made by the Philadelphia courts.]

The United States Circuit Court of Appeals, in the case of Mahler vs. Animarium,[2] declares against the issuing of a **patent of discovery** of a new fact or principle in the science of medicine.

The Supreme Court of Georgia, in the case of Minda vs. the State,[3] says that one accused of a criminal act **in order to sustain the defense of insanity** must prove that he was insane at the time of the commission of the crime. [In most criminal cases in which insanity is the plea of the defense the attempt to determine the mental condition of the individual is not made until some time after the commission of the act, when there is reason for the accused to simulate insanity. The decision of the Georgia Court more justly covers such cases.]

Falconbridge, Chief Justice of the King's Bench,[4] delivered a lengthy and valuable opinion on the important subject of **malpractice.** An action for malpractice was brought against two surgeons by a woman who had injured her foot, and who claimed that as a result of improper treatment the foot had become distorted and twisted, and that she was rendered permanently lame. It seems that the plaintiff called in the first defendant, but discharged him after he, together with another surgeon, had set the foot. The defendants claimed that the foot was properly set, and that the deformity and ensuing pain were the result of subsequent negligence and improper treatment on the part of the plaintiff. The defendants further claimed in their defense that proceedings against them had begun more than a year after the date of their last visit. The court stated that there is "no implied warranty on the part of a physician that he will effect a cure. . . . ; that when a surgeon treats a patient improperly, whether the service be gratis or not, he is liable to action; that when a patient by his own negligence aggravates his injuries, he cannot recover damages from a physician; that a medical man is liable when he neglects to give proper instructions as to the care and use of an injured limb." The patient claimed that the astragalus was dislocated and that there was no fracture of the fibula, and therefore an incorrect diagnosis had been made, offering in part proof of this the evidence shown by a skiagraph. The court believed that a fracture could not be absolutely thus diagnosed, to the extent, at least, of disclosing the line of fracture, and that after a lapse of two years little reliance could be placed upon a skiagraph. The evidence pointed to the conclusion that the unfortunate condition of the plaintiff was due to her own conduct.

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[1] Jour. Am. Med. Assoc., June 21, 1902, p. 1670.
[2] Jour. Am. Med. Assoc., 1902, p. 198.
[3] Jour. Am. Med. Assoc., Oct. 5, 1901, p. 936.
[4] Canad. Pract. and Rev., June, 1902.

Wilson[1] discusses the **medicolegal aspect of tuberculous joint disease,** and cites a number of legal cases bearing on this subject.

The Court of Appeals of Maryland[2] holds that **inaccurate conclusions** are apt to be reached when much weight is placed upon **expert testimony dependent entirely upon hypothesis.** It is not unusual to see two experts of equal ability come to diametrically opposite conclusions about a hypothetic case, and therefore the court holds that it is important to exercise unusual care in restricting this sort of evidence. [The "hypothetic question" is sometimes misleading and is often intended to obtain an opinion which the court would not otherwise permit to be given.]

William Lloyd[3] gives an interesting résumé of the **present attitude of the law to pseudomedical cults.** The medical law, he says, is sufficient to control those who falsely profess to be practitioners, and quacks, clairvoyants, and electropaths, who prescribe drugs, but it has no jurisdiction over those who, under the cloak of some cult, treat disease. The legislature does not interfere in those cases in which an individual selects for himself certain methods of treatment not recognized by the medical profession. The Court of Common Pleas of Pennsylvania held in a recent case that **osteopathy was not practising medicine,** according to the Act of May 18, 1893. Lloyd opposes repressive legislation, claiming that this would give these people an opportunity to pose as martyrs, and believes the solution of the difficulty to be found in education. [Repressive legislation other than that requiring all individuals who, whether for pecuniary reward or not, undertake to treat sick persons to pass the examinations of the State Board of Medical Examiners for license to practice seems to be inadvisable. It is futile to attempt to influence people by education either special or general, or both, because many of the people who are duped are above the average of intelligence of the community and are well informed of the views of sensible, scientific men of the absurdity of the claims of the "faddists."]

William Lloyd[4] discusses the question of the **liability of physicians who sign certificates of insanity.** The question which arises is: What are the liabilities of a physician who certifies that a person is insane when afterward that person proves to be sane? Any misstatement as to facts in such a certificate under the English Lunacy Act (53 Vict., C. 5, 317) renders one guilty of a misdemeanor. The statute in Illinois declares that a person is guilty of a misdemeanor who conspires unlawfully to commit a person to an insane asylum. However, if a physician is honest in his purpose but is mistaken in his diagnosis, what then is the extent of his liability? A physician is required to possess only the reasonable and ordinary qualifications of his profession, and must exercise reasonable and ordinary care, skill, and diligence, but beyond this he incurs no liability. The physician should observe the greatest caution in matters of this character, and should be warned against making insufficient personal examinations of the patient.

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[1] Amer. Med., July 6, 1901, p. 24.

[2] Jour. Med. Assoc., 1901.

[3] Phila. Med. Jour., June, 1902, p. 85.

[4] Phila. Med. Jour., 1902, p. 51.

The Supreme Court of Mississippi, in the case of The Cumberland Telephone Co. vs. Pitchford,[1] cut down the verdict of $25,000 to $12,500 in the case of a man who died 2 weeks after a fall to the ground as a result of coming in **contact with a live wire** while working on a telegraph pole.

The Supreme Court of Alabama, in the case of The Southern Railroad Co. vs. Crowder,[2] gave a verdict of **$15,000 damages** to a passenger hurt by a violent shock of a car, resulting in a hip injury and consequent **shortening and stiffening of the leg.**

The Second Appellate Division of the Supreme Court of New York[3] **does not think that a verdict of $20,000 damages is excessive** for injuries to a child 3 years of age, who after the accident suffered from complete paralysis and gradual wasting of one arm, possible impairment of the use of the legs, and probably permanent impairment of mental faculties, all of which would probably result in the child becoming a constant charge and source of expense.

The Court of Lyons[4] rendered an opinion against the parents of a child suffering from hereditary syphilis who **contaminated the wet-nurse.**

The First Appellate Division of the Supreme Court of New York, in the case of Williams vs. Underhill,[5] decided that damages could be recovered for **injuries resulting from fright,** provided they emanate from wilful wrongdoing.

The Supreme Court Jury of New York,[6] in the case of Asch vs. Goldsmith, decided that a physician had the **privilege of raising** [increasing] **his fees** when, by reason of his moving away from the residence of a patient, he was obliged to travel out of town to continue his attendance upon the patient.

Vaccination or Filth?—Mrs. Bridget Nugent[7] brought suit against Dr. Harvey Righter to recover for the death of her child, 6 years old. The case was tried before Judge McCarthy in Common Pleas Court No. 3, Philadelphia. The attorney for the plaintiff was C. A. Beasley, Esq., an antivaccinationist. It was alleged by the plaintiff that Dr. Righter used impure virus in vaccinating the child of Mrs. Nugent in September, 1900, and as a result of his careless method impetigo contagiosa developed, finally resulting in the death of the child on October 20, 1900. It was also alleged that in the treatment of the latter disease the defendant did not exercise proper skill and diligence. It was shown by the defendant that he had used more than ordinary care in vaccination; that it was impossible that the impetigo, which developed about a month after vaccination, and after the vaccination wound had healed, could have been introduced at the time of the vaccination. It was shown that the period of incubation for this disease is at most a few days. It was further shown that Dr. Righter had recognized the disease, had treated it properly, and had exercised unusual care in the management of the case. The testimony of several physicians seemed to show that the hygienic condition of the plaintiff's home was bad. Several witnesses for the plaintiff testified to the good hygienic surroundings. The jury awarded $1000 damages to the plaintiff. This is believed by many of the profession and by medical societies to be an unjust decision, and a number of the members of the profession intend taking an active part in continuing the case in the higher courts.

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[1] Jour. Am. Med. Assoc., Sept. 14, 1901.

[2] Jour. Am. Med. Assoc., Nov., 1901

[3] Jour. Am. Med. Assoc., Aug. 3, 1901.

[4] Gaz. Hebdom., Dec., 1901, p. 576.

[5] Jour. Am. Med. Assoc., Sept. 28, 1901.

[6] N. Y. Evening Post, Nov., 1902.

[7] Jour. Am. Med. Assoc., Mar., 1902.

X-RAY IN FORENSIC MEDICINE.

The medicolegal standpoint of the x-ray is discussed at some length by Dr. Leonard.[1] He makes a strong plea against the use of the x-ray by any save members of the medical profession who have received a special training in this line of work. A medical education is absolutely essential in this method of diagnosis. Leonard quotes liberally the paper of Golding-Bird entitled "Remarks on Skiagraphy and Fractures, Especially in Medicolegal Relations." Golding-Bird believes that this form of diagnosis is unnecessary in a large proportion of the cases, and advises its use especially in the doubtful cases, which he divides into two kinds: One in which the exact location or direction of a fracture is in doubt, or where a joint is involved; the other including the doubtful cases in which, though the doubt may not necessarily alter the treatment, yet which, should the patient later get a skiagraph on his own account, might place the surgeon in an awkward position, to say the least. Leonard takes exception to these views, and seems to be clear that every known means of diagnosis should be used in every case. Referring to the cases of Pott's disease and hip-joint fracture, Golding-Bird says that no surgeon could be held blameless who would not use the x-ray when a shadow of doubt existed as to the diagnosis between these two conditions. Leonard relies upon this method for obtaining correct detail and more accurate knowledge than is afforded by any other method of diagnosis. "The value and accuracy of the Röntgen ray in medicolegal cases is entirely dependent upon the experience and professional skill of the expert who made the skiagraph and translated it." He believes that the errors attributed to this form of diagnosis result entirely from the ignorance and inexperience of the operator. He quotes the opinion of von Bergmann, of Berlin, who recognizes the importance of the Röntgen method of diagnosis in the treatment of fractures. This method does not recognize an injury to the soft structures. "Symmetry in outline of the bones is not, therefore, to be demanded, and does not form any criterion of the efficiency of the treatment or of the extent of the total injury that was received." With the inexperienced, fractures may be overlooked in this method. It is impossible to determine the persistence of nonunion unless there is some displacement of the fragments that may be detected by the skiagraph. The author advises that the place should be identified by using lead letters placed upon the limb in such a manner that their shadows are cast upon the plate. The time and length of exposure and other data should be written in ink upon the plate. Skiagraphs may be introduced as evidence in cases of accident to determine the presence or absence of fracture, the extent of an injury to bones or their displacement, and to establish whether or not proper treatment was employed in cases of malpractice. He quotes a decision of the court,[1] as follows: "It is within the discretion of the trial judge to admit in evidence an x-ray photograph; and his determination of whether it is sufficiently verified, appears to be representative of the object portrayed, and may be useful to the jury, is not open to exception." Evidence should only be given by expert, reliable professional men.

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[1] Amer. Med., Jan. 25, 1902, p. 145.

MISCELLANEOUS.

Alden[2] describes the present method employed in the United States Army for the **personal identification of the soldier.** The object of this method is to prevent the re-enlistment of men who have been dishonorably discharged or who have deserted. He first describes the Bertillon system, acquaintance with which he deems necessary to the understanding of the present method employed by the army. This system was not accepted by the United States because it suggested, in the minds of the public, some association with the police, and with the detection of criminals, and also because the apparatus necessary was too bulky for practical use. In 1888 the Surgeon-General of the Army issued a circular requiring thereafter the vaccination of all soldiers on the outer aspect of the left leg 4 inches below the head of the fibula, as a means of identification, but so many soldiers became thus temporarily disabled that the order was revoked in December, 1891. The following plan was then adopted: For every man who enlists or re-enlists a card is made out on which are noted his name, age, height, the color of the eyes and hair, the condition of the teeth, the location of scars, tatoos, moles, and birth-marks. These cards are classified so as to facilitate the identification of men enlisting. The results of this system have been encouraging. From July, 1890, to March 31, 1901, 2168 cases have been identified. In 1898 there were 347 identifications; in 1899, 480; in 1900, 358; and in the first 3 months of 1901, 135. This system has had the effect of reducing the number of desertions. In 1891 the desertions were reduced from 2344 of the previous year, to 1593.

Knopf[3] reports the case of Thomas O. Boden, a **consumptive Irish**

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[1] Jameson vs. Weld, 45 a, 299; 93 me. 345. American Digest, 1900 A. August to March.

[2] Boston M. and S. Jour., 1902, p. 514.

[3] Jour. Am. Med. Assoc., 1902.

immigrant. In June, 1901, the Superintendent of Immigration issued an order debarring from all ports of the United States immigrants suffering with tuberculosis. As a result of this, Boden, an Irish immigrant, was detained by the immigration authorities because he showed signs of tuberculosis. In the discussion which followed Knopf claims that there is not sufficient evidence at present to warrant classifying pulmonary tuberculosis among the dangerous contagious diseases. He cites the opinion of Biggs and T. Mitchell Prudden, who hold that pulmonary tuberculosis is a communicable but not a contagious disease. If pauper immigrants suffering from tuberculosis are excluded from American ports, it may be possible that wealthy invalids suffering from tuberculosis may no longer expect the hospitality of foreign health-resorts. He concludes that this law is founded upon unscientific bases, and will result, if it is carried out, in great injustice to the individual as well as to the sociologic condition of the country. He hopes that the Supreme Court may take a different view of the matter and refuse to stamp the tubercular individual as hopelessly ill, or as suffering from a dangerous contagious disease.

The forensic aspect of **pseudohermaphrodism** is discussed in an editorial in "Medicine," March, 1902. Under the Roman law a hermaphrodite could be a possible witness to a will, and it was presumed that the hermaphrodite was a male. In the United States medicolegal questions arise regarding suffrage, marriage, divorce, sterility, and impotence. The presence of menstruation is not a proof of femininity. Cases of menstruation in the male have been reported by Pinel, Fournier, Forel, King, Rayman, and others. A tendency to this abnormality occurs in families in rare cases. While applying the term hermaphrodite in contempt to a normal person implies odium, it cannot be held to be a libel.

TOXICOLOGY.

Commercialism in connection with the manufacture and use of antitoxin and vaccine virus was discussed by Dalton.[1] Attention is particularly called to the distrust which necessarily arises in the public mind with the two important agencies, vaccination and antidiphtheritic serum. Dalton believes that the responsibility for the various fatalities which follow the application of these remedies should rest (1) with the manufacturer of the antitoxin and vaccine virus; (2) with the physician; (3) with the parents and guardians of the unfortunate victims. He blames the St. Louis Board of Health for careless and inefficient methods. In deciding the question of the source of the tetanus, it is necessary to inquire into the quality of the vaccine, the technic of the operation, the character of the lesion, and the manner in which it was treated. He concludes that the lesson to be learned from these catastrophes is the necessity of removing commercialism from matters important to the public health. Boards of health should examine into the quality of the drugs and prophylactic agents, and should be careful not to use any preparations that are not absolutely reliable. They should surround the manufacture and sale of such articles as antitoxin and vaccine virus with the strictest care. "To be impartial, boards of health must go out of business."

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[1] N. Y. Med. Jour., Dec., 1901, p. 1150.

Sheldon[1] cites a case of **gangrene from the use of carbolic acid** occurring in a colored man who applied a dilute solution of the drug to a sore upon his right foot. He accidentally spilled some of the acid upon his left foot, but none upon his right foot. This resulted in a gangrenous condition which necessitated, first, an amputation at the middle of the leg, and, secondly, as a result of the extension of moist gangrene, amputation through the middle third of the thigh. The application of the carbolic acid was the only explanation that could be given for the development of the gangrene.

Brown[2] described a fatal case of **acetanilid-poisoning** after the ingestion of 1 gram of acetanilid in divided doses for headache. The patient, 37 years of age, became cyanotic and slightly jaundiced. The temperature rose to 100⅖° F.; the pulse was 78. The urine was of a deep red color, almost black, and measured 300 cc. Subsequently the urine was completely suppressed. The color of the urine was found to be due to hematoporphyrin. The reflexes were at first increased, but finally disappeared. Examination of the blood from the fifth to the eighth day of the illness revealed the following condition: Hemoglobin, 40 % to 32 %; erythrocytes were from 2,200,000 to 1,166,000. The nucleated reds were slightly above 20,000, and the leukocytes were between 60,000 and 70,000. The patient died in 8 days.

Clegg[3] cites a case of **poisoning with oil of cedar** in a woman who took 90 minims of this drug for the purpose of inducing abortion. In half an hour she was comatose and suffered from violent epileptiform convulsions every 10 minutes. The pupils were slightly dilated and did not react to light. The face was flushed, the extremities cold, respirations hurried and irregular, and the pulse slow and full. An emetic was given and the stomach washed out. The patient recovered in about 3 days. The drug produced no effect upon the uterus.

Brumitt[4] cites a case of **poisoning by mercuric iodid,** of which ¼ of a grain had been taken. Poisoning with this mercurial salt is of rare occurrence.

Friedländer[5] reports 50 cases of **poisoning by stramonium** which have occurred in the last 30 years. The seeds and the fresh plants were commonly used.

Reynolds[6] gives a report of three old **epidemics of arsenic-poisoning.** The first occurred in Paris in 1828. The symptoms were chronic erythema of the hands and feet, paralysis of the legs and arms accompanied by gastrointestinal disturbances, and irritation of the eyelids and bronchial tubes. The second epidemic was of Hyeres, in 1888, and was due to contamination of wine by arsenic which was used by mistake instead of potassium sulfate employed in the process of "plastering wines." The symptoms presented in this epidemic were fever, congestion of the conjunctivas, edema of the eyelids, and gastrointestinal disturbances. There were 435 victims, of whom 11 died. The third epidemic occurred in Havre, and was the result of homicidal poisoning. Of the 15 persons affected, 3 died. The symptoms consisted in disturbances of digestion, catarrh of the larynx and bronchial tubes with various eruptions, and sensory disturbances and palsies.

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[1] Med. Rec., v, 60, No. 20, p. 773, Nov. 16, 1901.

[2] Am. Jour. Med. Sci., v, 122, No. 7, p. 770, Dec., 1901.

[3] Phila. Med. Jour., Nov. 8, No. 22, p. 964, Nov. 30, 1901.

[4] Australasian Med. Jour., vol. xxii, No. 11, p. 476.

[5] Berl. klin. Woch., Mar., 1901.

[6] Med. Chron., April, 1901.

Menasses[1] records 2 cases of **poisoning from the external application of acetanilid.** In the first case it was applied for eczema of the buttocks. In 24 hours the child was cyanotic, temperature was subnormal, the pupils were dilated, the heart weak and rapid, respirations shallow and feeble, and the extremities cold. In the second case the drug was applied for a severe scald of the buttocks. On the second day there was every evidence of prostration and cyanosis. Neither case was fatal.

St. Clair Thomson[2] describes a **case of anilin-oil poisoning.** Anilin oil, a 10 % aqueous solution of cocain and rectified spirits in equal volumes, was given for the relief of furunculosis. This was applied on a pledget of cotton at bedtime. In 2½ hours there followed symptoms of cyanosis with rapid and small pulse. All the symptoms subsided after the discontinuance of the drugs.

Two cases of **fusel-oil poisoning are reported** by Futcher.[3] Both patients drank a considerable quantity of fusel oil. They remained unconscious for 8 hours. The urinary findings were interesting. The urine was smoky, possessed a fruity, aromatic odor, was acid in reaction, and had a specific gravity of 1025. It responded to the tests for albumin and reduced Fehling's solution. There was, however, no rotation of the rays of polarized light in one case, either to the right or left, but in the second case the rays of light were rotated to the left. Microscopically there were numerous granular casts present. The urine was normal 3 days later. Experiments upon dogs resulted in the discovery that when fusel oil was administered it caused the elimination of glycuronic acid by the urine which has the property of reducing Fehling's solution.

Monks[4] reports a case of **poisoning by dinitrobenzene.** This drug is not volatile at ordinary temperatures, but when warmed gives off fumes which cause rapid intoxication. Eight grains is said to be the fatal dose for an adult. A man had taken home some roburite which his wife mixed with some insect-powder and sprinkled it over the floor and beds. In the early morning one of the children awoke complaining of pain in the abdomen, which was later followed by vomiting and diarrhea. In the next few hours the other members of the family were seized with headache, malaise, and pain in the stomach. When some hours later they were taken to the hospital, they were cyanosed, the respirations were quick and shallow, and the pulse rapid and soft. The mental faculties were dulled, and the urine was dark colored, but contained no albumin or sugar. The symptoms all disappeared in 24 hours. The poisoning was due to the fact that when the roburite was brought into contact with the skin when the family were in bed, the heat of the body volatilized the dinitrobenzene of the roburite, which disseminated itself in the air of the room.

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[1] Internat. Med. Mag., 1901.

[2] Lancet, April 20, 1901.

[3] Amer. Med., Aug. 10, 1901.

[4] Lancet, v, 162, 1902, p. 89.

Racine[1] cites a case of **fatal poisoning** by the **ingestion of benzine.** A child of 10, after drinking 10 to 15 grams of benzine, lost consciousness, became cyanosed, had clonic convulsions, dilated pupils, lowered pulse and respirations, and died in 2 hours. Postmortem, the blood was fluid, bright red, and there were numerous hemorrhages in the kidneys, lungs, liver, and the mucous membrane of the intestines. The gastrointestinal tract and the mucous membranes of the respiratory tract were inflamed and the brain was hyperemic. The kidneys were inflamed and the urine was suppressed.

Kemper[2] discusses the question of what constitutes **septic poisoning in accident policies,** and concludes that the surgeon is entitled to an indemnity when by reason of a wound he is disabled from practising his profession, whether or not septic poisoning ensues, or he is entitled to indemnity if an old wound or a new wound is infected during the operation. If he cannot recover from an infection of a sore or abrasion, then he receives no benefit from the clause attached to his general policy providing for septic poisoning.

Leffmann[3] reviews **the Haines case,** and makes some important **medicolegal deductions** regarding **poisoning by arsenic.** In recent years the use of embalming fluids containing compounds of arsenic has increased the difficulty in examining the viscera postmortem for the presence of arsenic in suspected cases of poisoning. Unfortunately the fluids used by undertakers vary in composition, and an undertaker may use one syringe for a number of different fluids without intervening cleaning. Thus arsenically free fluids may be contaminated by a syringe that had previously been used for a fluid containing arsenic. While arsenic is known to be an irritating poison, there are a few examples of deaths apparently due to this drug which have not been attended by inflammatory conditions. [It may be possible that, on the ingestion in toxic quantity of arsenic in combination as a soluble arsenite, death may occur before inflammation of the stomach may be produced and thus the necropsy may not reveal an inflammation.] He refers to the chemical analogy existing between arsenic and phosphorus, and, therefore, the anatomic changes produced by them may be similar. Fatty degeneration of the viscera has been found in cases of poisoning by both substances. Here, he says, we have a pathologic symptom which may lead to important deductions even when there is no evidence of local irritation of the stomach. [This condition would not apply to most cases of death due to the administration of arsenic, because in such cases death occurs so soon after the administration of the poison that any degeneration of the fats of the viscera would probably be so slight as to escape observation or to render it doubtful as one of the characteristics of acute arsenical poisoning.] However, he states that it is important to go further than making chemical tests and the inspection of the viscera in cases of supposed arsenical poisoning. In the case of Mrs. Haines's child one of the examinations of the viscera was made after burial, so that the question of postmortem imbibition arose. By experimentation this has been shown to be possible. In the case under discussion there were found 17 grains of arsenic in solution and in the tissues of the different organs. Some fluid had been used in embalming, but it was alleged that it was nonarsenical. [It was ascertained subsequently that the embalmer was not certain that the syringe used in making the injections did not contain some arsenical embalming fluid employed in making an injection of a body prior to its use in injecting the Haines child.] Embalming fluids have been shown to contain arsenic, zinc, formaldehyd, hydrogen dioxid, and sulfites, while glycerin is used in nearly all of them. The fact that zinc is present in some of the arsenical preparations could be an aid in the methods of discovering the presence of arsenic in the fluid. He cites an example illustrating this point. He suggests that official note should be taken of the modern methods of undertakers, and that there should be some regulation of the use of the arsenical fluids. He believes that each type of fluid should be "keyed," that is to say, it should contain a small amount of some substance readily identified by simple tests. He believes that it cannot now be claimed that 2 grains of arsenic will occasion death in an adult, nor that the mere finding of arsenic in the abdominal viscera necessarily proves the antemortem introduction of the poison.

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[1] Vierteljahr. f. gerichtl. Med., 1901. [2] Phila. Med. Jour., Feb. 15, 1902, p. 328.
[3] Phila. Med. Jour., April 19, 1902, p. 704.

Laschtschenkow[1] refers to the **poisoning of 200 persons** in Charkow, by **cream-tarts** which were made of milk, butter, eggs, sugar, spice, nuts, and rum. One case resembled arsenical poisoning. Most of them were not very severe. Examination of the pots and the substances used in making the tarts showed no contamination with metallic poison. Bacteriologic examination was made of the cream used and colonies were grown which resembled Staphylococcus pyogenes. The isolated coccus injected into animals was unusually pathogenic. The presence of this coccus in the tarts was explained by the fact that the cakes had spoiled before they were sold.

MEDICOLEGAL TESTS.

Diagnosis of the Origin of Blood-stains.—Wood[2] discusses the methods of the examination of blood-stains for medicolegal purposes. The preliminary examination consists in observing the gross appearance of the stain. It is important also to observe the color of the stain which reacts in a definite manner to light and air. A blood-stain grows darker for a period of 10 days when exposed to light and air. When exposed to direct sunlight, it quickly changes to deep brown. The form of the stain is important, as showing the direction from which the blood came. For example, if a drop of blood strikes upon a smooth surface at a more or less acute angle, it assumes the shape of a pear. If it strikes a vertical surface, the bulk of the blood will gravitate to the lower part of the stain. If it falls upon a rough surface, it coagulates so quickly that the bulk of blood remains at the stem-end of the pear-shaped stain. If it strikes at right angles, the stain is usually round. It is important to note whether or not the stain is dry. Experimentally it takes a drop of blood 2 hours to dry in a room at ordinary temperature and under ordinary atmospheric conditions. Wood resorts to three methods in the systematic examination of blood-stains for medicolegal purposes. First, the chemical tests; (2) the optic method; (3) the microscopic examination for the inspection of red blood-cells. Of the chemical tests, the most important, he considers, are the guaiacum test, the hemin test, and the sodium tungstate test. The so-called optical method is the spectroscopic test, which has the disadvantage of requiring a considerable volume of blood. The microscopic examination distinguishes the kind of blood; that is, whether it comes from a mammal, bird, fish, or reptile. Human blood may be distinguished from the blood of other animals, except that of the camel family, by measuring the diameter of the red blood-cell. If the average diameter of the red blood-cells is $\frac{1}{3200}$ to $\frac{1}{3300}$ of an inch, the probability is that they are from human blood. If the average diameter is less than $\frac{1}{4000}$ of an inch, the chances are that the corpuscles are not from human blood. In conclusion, he briefly refers to the agglutination test.

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[1] Aerztl. Sachverstandigen-Ztg., Nos. 9 and 10, 1901.

[2] Boston M. and S. Jour., Nov. 19, 1901.

Scheppegrell[1] calls attention to the importance of careful **ear examinations from the medicolegal standpoint.** He cites the following case: The patient, a colored woman, had 8 days previously, by the advice of a neighbor, placed a mixture of salt and cayenne pepper in her ear for the relief of toothache. A few days later an examination revealed the presence of a perforation and a purulent discharge. Upon questioning the patient it was discovered that she had used an old toothpick to remove the pepper and salt, and while so doing she had felt a sharp pain, and at once had desisted. In the mean time the patient had sworn out an affidavit against the neighbor, alleging that the advice was given with malicious intent, and that the conditions which arose, thereby threatening her hearing and health, were the results of the irritating substances inserted into her ear. The case came to trial, but was immediately discharged when Scheppegrell testified to the opinion that the perforation was due to the insertion of the toothpick. The author calls attention to the importance in medicolegal cases of eliminating all conditions that may have existed before the accident to which deafness may be attributed.

Linossier and Lemoine[2] claim to have established that the so-called **specific precipitating serum for human blood** precipitates also the serum of cows, horses, dogs, sheep, pigs, guinea-pigs, and chickens, but that the reaction to human blood is incomparably more sensitive. To avoid error thereby made possible, the reaction should be practised only

[1] Amer. Med., June 28, 1902.

[2] Gaz. Hebd. de Méd. et de Chir., No. 26, 1902, p. 297.

upon diluted solutions of blood. For example, a solution of one-thousandth dilution seems always to be precipitated by serums of a corresponding activity. The authors claim in this way to make distinctions otherwise impossible.

An excellent review of Lacassagne's book, "Vade Mecum du Médecin Expert," on medicolegal examination of **blood-stains and of the spermatic fluid** appeared in the "Journal de Médecin de Paris," Aug. 11, 1901. He states that there are four qualities in a stain which prove the presence of blood: (*a*) the ozonoscopic reaction of Van Deen, (*b*) the hemin crystals, (*c*) the spectroscopic reaction, (*d*) the examination for red blood-cells. Among other tests given, proving the presence of blood, is the test for crystals of hemin. The spectroscope is employed to determine the presence of oxyhemoglobin and methemoglobin. Oxyhemoglobin gives two bands between D and E, the first narrow and shaded, the second much larger, clearer, and flatter. When ammonium sulfid is added, the two bands disappear, and instead there appears a large, feeble, slightly shaded band of hemoglobin. The spectrum of methemoglobin is always superimposed upon the spectrum of oxyhemoglobin: a narrow dull band situated between C and D, a little nearer to C. The spectrum of blood treated by the cyanids resembles that of hemoglobin. Upon adding ammonium sulfid there appear two clear narrow bands, the first deeper and more shaded between D and E, the second flat and more feeble at E. These four spectra, he says, taken two by two, are absolutely specific. The spectrum of hemin in acid solution is a single band between C and D, very near to C. In alkaline solution it is a single band much nearer to D, and occupying almost all of orange. In determining the origin of blood it is important to measure the diameter of the red corpuscles. In mammals they are circular and in other classes elliptic. The diameters in millimeters of the red corpuscles are as follows:

Man	0.0077	Hog	0.0062	Cat	0.0056
Dog	0.0070	Ox	0.0058	Sheep	0.0045
Rabbit	0.0064	Horse	0.0057	Goat	0.0040

It is necessary to count 100 of the blood-corpuscles to determine their diameter. He concludes that the blood is not human if the reaction of Van Deen is not obtained; it probably is human blood if it is obtained, and certainly if the spectra two by two are obtained. If the characteristic corpuscle is isolated, or if crystals of hemin are obtained, one of the last three signs suffices to make a positive diagnosis. To distinguish the stains of spermatic fluid the (*a*) chemical and physical character of the stain is studied, and (*b*) a microscopic examination is made.

Baker[1] reports some interesting details concerning **the Erickson murder.** The murderer was brought to justice in part by the **recognition of blood found upon his shoes** and clothing and upon a piece of woodwork. This was in spite of the fact that the shoes had been blackened repeatedly and worn several days.

Wood[2] reviews the history of **the serum test for blood,** citing the work of Krause, Bordet, Tchistovitch, Uhlenhuth, Wassermann, Schutze, Meyers, Nuttall and Stern. [In the American YEAR-BOOK of 1902 this work is excellently reviewed in detail.]

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[1] Boston M. and S. Jour., Nov. 7, 1901.

[2] Boston M. and S. Jour., 1901.

Whitney[1] describes the **technic employed in the making of the serum test in rabbits.**

Nuttall and Dinkelspiel[2] have studied the **specific anti-bodies** in the **blood of animals** treated with different serums, and refer to their possible use in forensic medicine. They injected rabbits with blood, pleuritic exudate, etc., 5 cc. to 10 cc. being injected into the peritoneum in gradually increasing doses. After the fifth or sixth injection they obtained an effective anti-serum. They also injected rabbits with the serum, defibrinated blood, and pleuritic exudate of dogs, sheep, oxen, horses, and cats. In their conclusions they confirm the recent discoveries of specific precipitates in the blood of animals so treated. While these precipitates may produce a slight reaction with the serum of allied animals, they believe that they are specific. The substances in the serum causing the formation of these bodies are remarkably stable.

An editorial comment appears in the "Journal de Médecin de Paris," June 8, 1902, upon the interesting **experiments of Nobele,** designed to confirm the results of Wassermann and Schütze. He injected 10 cc. of ascitic fluid taken from a woman suffering from uterine tumor into the peritoneal cavity of a rabbit. After 6 injections the animal was killed and its serum, mixed in equal parts with human serum, produced a flocculent precipitate. In another experiment a rabbit was injected subcutaneously with 10 cc. of a liquid which was expressed from a fresh human placenta. After 6 injections the animal was killed and its serum reacted upon human serum. Nobele dissolved dried blood in physiologic solution of sodium chlorid, or a weak solution of caustic soda. He demonstrated that the serum of rabbits produced no reaction with the blood of dogs, horses, oxen, sheep, rabbits, pigs, and cows. The serum of an injected rabbit precipitated the serum of human milk, pus, saliva, nasal secretions in coryza, and albuminous urine. Putrified blood reacted equally well. The stain of human blood made upon linen many days, up to 2 months, previously, when treated with a solution of sodium chlorid gave the characteristic reaction. A piece of linen stained with human blood was washed with water and then dried so that the stain was distinguished only with great difficulty. The linen was plunged into a solution of sodium chlorid, and when the serum of the injected rabbit was added there followed a feeble precipitation. Linen saturated with human blood in 1893 was treated in the same way, but gave no reaction. Rusty scissors stained with human blood 2 months previously were scraped, the debris taken up in a solution of sodium chlorid, and to this mixture was added the serum of an injected rabbit. The characteristic reaction followed. A few drops of blood were placed upon 3 pieces of glass and subjected to temperatures of 75°, 100°, and 125° C. respectively. The stains were then dissolved in solution of sodium chlorid and it was found that the characteristic reaction failed only in the specimen subjected to a temperature of 125° C. Nobele gave to rabbits by the mouth ascitic fluid from a woman suffering from uterine tumor, but found that the serums of such animals did not react to human serum. One must then conclude that digestion destroys the substance which produces in serum this property of precipitating human blood-serum. Nobele desiccated the reactive serum in vacuum and obtained thus a substance which he was able to preserve when it was hermetically sealed. He also was able to preserve a serum which displayed activity at the end of 6 months.

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[1] Boston M. and S. Jour., April 24, 1902, p. 429.

[2] Jour. of Hygiene, July, 1901.

Symanski[1] concludes from a **study of blood and fragments of tissue** from the body of a patient who had succumbed to **traumatic tetanus** 5 weeks before, and whose body had been exhumed for the purpose of confirming the diagnosis, that it was possible to demonstrate the presence of tetanus toxin in the body even after putrefaction had taken place. The tissue was passed through a porcelain filter, and the red filtrate was injected into white mice in quantities varying from 0.25 cc. to 1 cc. Some of the mice lived for 4 days, manifesting the symptoms of tetanus. Most of them succumbed to this disease, but one recovered. Symanski draws his conclusions from these facts alone.

MENTAL CONDITIONS.

Thayer[2] discusses the **responsibility of murderers of unsound mind.** He reports the cases of 5 Maine "murders," and believes that the following conclusions may be reached in the present conditions in the State of Maine: "That insanity has never been a cloak for homicide. That within the last 25 years there have been at least 5 cases of homicide . . . . in which the doer of the act has been insane before and after he did the act, and in which there is at least a high degree of probability that the act itself was the product of his insanity. That these men have all been convicted of murder. That the convictions are to be attributed in part to the explicit, lucid, archaic, and vigorous rulings of the Maine courts. That the practical working in Maine of the only proper legal criterion is to concede irresponsibility only to idiots and maniacs. That a realization of this fact on the part of the courts, prosecuting officers, and the public, together with increasingly accurate knowledge of the nature of mental disease, must inevitably lead to verdicts which are at the same time more just, more scientific, and more humane."

McDonald[3] reports a full account of the **trial, execution, autopsy, and mental status of Leon Czolgosz.** On September 6, 1901, Czolgosz shot President McKinley at Buffalo, New York. The legal question to be decided was: Did he know the nature and quality of the act that he was doing, and that it was wrong? If so, then he was legally responsible for the act. From the medical standpoint the question was: Was the prisoner insane at the time that he committed the crime? The trial took place on September 24, 1901, in the city of Buffalo, the Commonwealth being represented by the Honorable Thomas Penny, District Attorney, and Mr. Heller, his assistant. He was defended by Loran L. Lewis, and Robert Titus, of Buffalo. Czolgosz was examined by Drs. Joseph Fowler, Floyd T. Crego, and James W. Putnam, in order to determine his mental condition. Subsequently Drs. Heard and McDonald also examined into the mental condition of the prisoner. He was electrocuted on Thursday, October 29th, at Auburn prison. The autopsy, made by Mr. Edward A. Spitzka under the direction of the physicians, revealed the absence of any signs pointing to disease of any of the organs, including the brain, which was normal in all respects. The microscopic examination was not made, as the warden of the prison declined to permit any portion of the body to be removed from the prison. The result of the examination into the mental condition of the criminal resulted in the conclusion that he was sane. There was nothing about the physical condition to indicate any form of mental disease or degeneracy. No illusion or hallucinations could be developed. He was not "inspired," nor had he any "mission to perform." It may be claimed that the statement made by Czolgosz that "the President was an enemy to good working-people" was a delusion, and, in the broadest sense of the term, it was. That is to say, it was a false belief, but not one due to disease of the brain, but rather the result of an ignorant mind, of faulty education, and of warped mind and judgment. Czolgosz was a self-confessed anarchist, a believer in the principles of "free society," as they are taught by Emma Goldman. The motive for the crime is found in these beliefs. In the same paper Edward Spitzka gives a detailed report of the postmortem examination of Czolgosz, including numerous measurements of the skull, brain, thorax, and abdomen. A full and exhaustive study of the body was impossible on account of the peculiar conditions imposed by the prison warden, who cut the examination short in his anxiety to dispose of the body.

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[1] Centralbl. f. Bact., No. 25, p. 976.
[2] Boston M. and S. Jour., Feb. 22, 1902.
[3] Am. Jour. of Insanity, Jan., 1902, p. 369.

Wiglesworth[1] reports the case of a **murder,** the **result of pure homicidal impulse.** The patient was an inmate of an asylum, who assaulted an inoffensive dement. The deed resulted from a homicidal impulse, and not a delusion. The patient had stated to the matron upon the morning of the crime that she had an uncontrollable desire to kill some one. The important question which arose was: "Was the woman at the time when she committed the crime suffering from any disease which affected her mind, which prevented her knowing the nature and quality of the deed, and did she know that the act was wrong?" The answer to the first was negative; and to the latter, that she acted in response to an impulse which cannot be considered responsible.

Mills[2] divides **criminal assassins** into 4 classes: (1) Sane conspirators; (2) assassins recognized as insane; (3) degenerates who are not insane; (4) degenerates of doubtful sanity. He classes the Orloffs, the assassins of Peter the Third of Russia, the assassins of Paul the First of Russia, of Gustavus of Sweden, and of President Lincoln, as sane.

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[1] Jour. of Mental Science, April, 1901.

[2] Phila. Med. Jour., Oct. 26, 1901.

Ravallac, Louvel, and Guiteau are classed as insane. The assassins of Marat, Garnot, Canovas, the Empress of Austria, and King Humbert are placed in the third and fourth classes. He believes that Czolgosz was sane and free from any marked signs of degeneracy.

Smith[1] describes a case of **epileptic homicide.** The accused had killed his wife and child. It was claimed that he was unconscious at the time. No motive for the crime was assigned, and his previous good character and honorable service in the army were introduced as evidence. A number of his relatives were epileptics. He was acquitted on the ground of insanity.

Stern,[2] in discussing **alcoholism and crime,** makes some interesting suggestions as to the disposition of the criminal alcoholic. It is conceded, he says, that 70 % of all perpetrated crimes is directly or indirectly the result of alcoholism. He shows how the alcoholic habit changes the character, lowers the standard of morals, weakens the mind, and destroys the finer nature of man, and points out also the evil effects upon the offspring. He claims that the prison is the wrong place for the inebriate, that he there sinks into a confirmed criminal. Just as prophylaxis of disease should be studied, so should prevention of crime, and the drunkard should be treated before he commits a crime. There should be attached to every police court a medical officer who should class the inebriates into (*a*) occasional offenders, and (*b*) confirmed alcoholics. They should be sent to a **public reformatory,** the expenses of which should be defrayed by the income of the liquor tax. Thus the criminal alcoholics may be converted into useful members of society.

Schram[3] discusses the **medicolegal aspects of cerebral apoplexy,** especially in its relation to testamentary capacity. He outlines a method of examination to discover the mental capacity of a patient suffering from apoplexy. He cites Gallard in this connection, who states that an aphasic is "(1) irresponsible if he cannot manifest his lucidity by words, pantomime, or writing; (2) his intelligence is impaired if he manifests his lucidity by writing incompletely or sluggishly; (3) he is responsible if he manifests his lucidity clearly and intelligently by words, pantomime, or writing." An aphasic is competent to testify if he comes under the last two headings. An examination should be made of written or spoken speech and gesture. Schram shows how this should be done. The family history should be carefully gone into. The author then proceeds to describe the apoplectic state, which he considers in three forms: (1) The comatose, (2) the postapoplectic state, and (3) the class with transient acute subjective symptoms, with partial or complete thrombosis, embolism, or hemorrhage occurring in the latent portion of the brain, in which the emotional disturbances are slight, and the intellect only slightly disturbed. He finally considers the mental status of each variety of aphasia.

At the Old Bailey[4] at the last session there were **two cases on trial**

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[1] Jour. of Mental Sciences, July, 1901.

[2] Amer. Med., Feb. 1, 1902, p. 177.

[3] Med. News, July 20, 1901, p. 90.

[4] Lancet, Nov. 15, 1902, p. 1344.

for murder to which the **defense of insanity** was brought. One was an epileptic who had killed his wife with a knife. His counsel endeavored to show that by reason of his mind's being weakened he exaggerated the danger of the attack made upon him by his wife, and in defending himself with a knife had taken her life. The jury found the prisoner guilty of manslaughter and sentenced him to 5 years' penal servitude. In the second case the prisoner had murdered his daughter, thus revenging himself on the mother, who he believed was unfaithful to him. The Justice summed up the case thus: "It is a case, you can see, of *video meliora proboque, deteriora sequor,* and I can't help myself." The prisoner was sentenced to death. The "Lancet" calls attention to the fact that, while in view of the evidence in this case it cannot be doubted that these prisoners were responsible for their actions, on the other hand, there have been instances in which prisoners whose mental condition was no worse than these, but who were better able to defend themselves, have secured a verdict of guilty but insane.

CRIMINALS AND SUICIDES.

Lacassagne and Martin[1] report the conclusions of the Congress of Criminal Anthropology of Amsterdam relating to the **influence of criminal anthropology upon the application of the law.** They concluded that among the individuals prosecuted as criminals are a number who were degenerates and of unstable equilibrium. These are more or less irresponsible for their acts, and cannot be properly called criminals. Then there is the true criminal, who has not the physical or psychic stigmas which permit of his being classed as irresponsible. They call attention to the importance of studying these individuals from a biologic standpoint, as well as the conditions under which the crime is committed. In the genesis of crime the law of imitation, the influence of religious ideas, the law itself, the different factors of the sociologic orders, all play important parts. The influence of alcoholism, tuberculosis, and syphilis upon physical degeneration is cited. They propose the creation of special asylums for the insane criminal and the extension of the medical service in prisons for the purpose of the careful observation of the criminal. The physician should influence the moral treatment of the criminal by education and work, and shall judge when the criminal is fit to be returned to society again. Special colonies should be created for this purpose. The present methods employed against criminals are imperfect, and we must look to prophylactic measures to prevent the progress of crime.

Weymann[2] describes a method for the **identification of criminals through the fundus of the eye.** He claims that identification can be secured by drawings of the papillas and a surrounding retinal circle distant from the scleral ring by two papillary diameters. The points to be considered are: "(1) The method of division; (2) the exact points of division; (3) the exact angle of the vessels with the primary meridians; (4) the exact distance of one divisional point from the other; (5) the relative size of the divided twigs; (6) the angle of division; (7) the course of the twigs; (8) the exact difference everywhere and at every point between venous and arterial branches." The only objection to this method is that it requires a great deal of time, and may be interfered with by clouding of the mediums.

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[1] Jour. de Méd. de Paris, Aug. 6, 1901, p. 405.
[2] Jour. Am. Med. Assoc., Feb. 22, 1902.

Suicide in France.[1]—In 1898 there were 9438 suicides, as against 9356 in 1897. Of these, 1537 were attributed to poverty and reverse of fortune; 943 to domestic troubles; 624 to love or jealousy; 1226 to drink; 2705 to various troubles, of which physical pain accounted for 1785; 1347 to cerebral disease, and 1056 to unknown causes.

Suicide in the United States.[2]—The total number of suicides for 1899 was 5340; for 1900, 6755; and in 1901, 7245. Of the latter, 5850 were males and 1395 were females, a proportion of 5 males to 1 female. Of professional men there were 33 physicians, 10 attorneys, 10 clergymen, 6 bankers, 6 journalists, and 1 college professor. The cause was in 2980 cases attributed to despondency, in 1643 cases to unknown causes, 674 to insanity, 618 to ill health, 541 to domestic infelicity, 439 to liquor, 283 to disappointed love, and 67 to business losses. Poisoning was the chief agency, accounting for 3106 cases. There were 2476 cases due to shooting; 614 to hanging; 513 to drowning; 356 to cutting of the throat, and the remainder to various other means.

Wright[3] reports an **unusual form of suicide.** A man 63 years of age slashed himself 3 times across the back of the neck with a razor. The first incision cut the skin and fascia, the second cut through the trapezii muscles, and the third entered the spinal canal between the atlas and axis, opening the theca vertebralis, and wounding slightly the posterior columns of the cord. All three incisions were made while the razor was held in the right hand. There was no autopsy made. Rapid loss of cerebrospinal fluid probably produced death.

In an editorial in "American Medicine"[4] the **prophylaxis of suicide is discussed.** Suicide occurs in those who are sick mentally, morally, and physically, or in all three ways. Reference is made to the tendency to suicide which runs in families, and a case is cited in which 22 suicides occurred in the same family in 50 years. Especial caution should therefore be used in those cases in which the family history shows a tendency to suicide. Attention of physicians and nurses is called to the necessity of suspecting the possibility of suicide to a greater extent than exists at the present time.

In an editorial in the "British Medical Journal"[5] it is stated that a study of the **annual report** of the Commission and Directors of Convict Prisons for the year 1901, ending March 31st, **shows a steady decrease in crime throughout England.** Since 1880 the number of prisoners committed to prison on indictment has declined 28.8 % absolutely, and 41.8 % relatively to the population, and the number of those committed summarily has decreased 25 % absolutely, and 22.5 % relatively to population. By indictment cases is meant serious crimes. Astor, of the Société Générale des Prisons, attributes the decrease in crime in England to the emigration of the "turbulent and dangerous individuals" and the "work of the preservation of young waifs and strays by private charitable effort." More weight must be given to the latter factor. Prison authorities believe that crime is best controlled by regulating youthful offenders. Of 50 youthful offenders recently discharged from convict prison, 11 were under 15 years of age and 23 were under 21 years of age. Juvenile offenders under 16 years of age receive special treatment, such as technical instruction, physical drills, general education, and work with rewards. It is hoped in this way to check crime in the young. The death-rate of prisoners is about half what it was 20 years ago. In 129 cases of insanity among prisoners, 91 were insane upon admission, and 21 of the remaining 38 developed insanity within a month of their admission. It is to be especially noted that as crime decreases there is a corresponding increase in the number of criminals showing signs of mental deficiency. These really do not belong in a prison; neither, on the other hand, is an insane asylum the proper place for them. Under the present system they receive special treatment, conspicuous in which is a system of reward.

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[1] Med. Rec., Jan., 1901.

[2] Med. Rec., Mar. 1, 1902.

[3] Lancet, Sept. 14, 1901, p. 744.

[4] March 22, 1902, p. 454.

[5] Oct. 5, 1901, p. 893.

Shufeldt,[1] reviewing **William Lee Howard's novel "The Perverts,"** attempts to point out the imperfections of the legal system of this country as illustrated in the method employed in the treatment of mental or sexual perverts; the necessity for studying those psychologically diseased; and to demonstrate the "curse that the Church has proved itself to be in all these things." Howard states in his novel that "continued immoral or dishonest acts of a man are only external expressions of the individual's diseased, distorted, or undeveloped physico-psychic organism. It is the organic, physiologic, matter-of-fact conditions existing through heredity plus the environments and education which determine the moral and immoral character of the individual, and any attempt to correct a pathologic condition through persuasive appeals to an unknown power is a humiliating exhibition of superstition and ignorance of nature's laws," and that "the control of criminals and the study of the prevention of crime should not be in the hands of the well-meaning but ignorant—in physico-psychic matters—religious bodies."

MEDICAL EDUCATION AND MEDICAL LICENSES.

[There is no more interesting subject in medicine than that which concerns itself with the methods employed in medical education, with questions of advanced standing, qualifications and examinations for licenses to practise medicine, and the reciprocity of licenses. In most schools the system of teaching the fundamental studies—that is, physiology, histology, chemistry, and anatomy—is faulty. In many schools these branches are usually not presented in an applied or correlative manner nor in pedagogic order. Commercialism is accused of being responsible for the graduation of men who lack knowledge of the first principles of medicine, and whose examination papers in State Board examinations show them to be illiterate to the last degree. There is a crying need for a system which shall provide for a national board of examiners, that shall hold examinations for medical practitioners who desire to practice in States other than those in which they are licensed. The standard and character of the examination should be such that every State in the Union shall recognize the certificates awarded by such a board to one successfully passing the examination, without sacrificing in any way the standard of medical education established by the individual State boards. The character and scope of the examinations must necessarily differ from those of the State examinations, which latter should be held for recent graduates. Every measure which tends to elevate the requirements of medical education should receive the earnest support of every practitioner of medicine.]

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[1] Pacific Med. Jour., 1902.

"Should the So-called Fundamental Branches of the Study of Dentistry and Medicine be Taught by the Same Faculty?" is the title of a paper read before the Pennsylvania State Dental Society, July 9, 1902, by Henry Beates, Jr., of Philadelphia. Beates claims that the **evolution of the professions of dentistry and medicine has been retarded** by unprincipled commercialism. Dental and medical education has fallen below the necessary requirements, as proved by the fact that laws have become necessary to govern their practice. "Obstructive commercialism" has been illustrated by those colleges that have made strenuous efforts to overcome the law requiring "adequate preliminary education" and efficient medical training. The enforcement of such laws affects the income of colleges of dentistry and medicine. He points out the ease with which students may be given advanced standing. Institutions have substituted work done in fundamentals in one branch of arts common in name with those of another, and students have taken advantage of this fact to gain short-cut diplomas. The "commercial professor" has endeavored to show that the fundamentals as taught in the high school or academy, in the biologic schools, or in general or technical colleges, were one and the same thing. It was found that students in pharmacy, dentistry, and medicine fell victims in large numbers to this commercial scheme, for the graduate who obtained his diploma through this short-cut method discovered, upon applying for examination, that he was ineligible. This was a great blow to the commercial medical college. Beates claims that the fundamentals as taught in colleges to-day are useless. As taught in the medical colleges at present general chemistry should be required for the matriculation examination, and the chemistry in the course should include only that phase of it which applies to the special art.

Henry Beates, Jr.,[1] says that medical education is actually standardized by examining and licensing boards In order fully to understand the subject it is necessary to review the methods of medical education prior to the era of legislative control. That so many incompetent physicians possess the right to practise medicine is explained by the fact that medical colleges have not been discriminating enough in the distribution of their diplomas. Acts of Assembly may control medical education by raising the standard of State examinations, making it essential that the final examinations of a medical college be of a sufficiently high grade to meet the requirements of the State examinations. If our laws are not achieving the results for which they were enacted, the reason must be ascertained. A recent graduate may be able to pass an examination because he has memorized certain facts, while a recognized authority would fail technically to pass a similar examination which deals more with abstract facts than the application of principles. So that as the medical law exists at present it really perpetuates the very evils it is designed to correct. The relations of general education to medicine must be considered in arranging the topics for examination. The scope of the examination will determine those subjects included in the course of study leading to the degree, while the character or type of the examination must fix that of the teaching. This will accomplish one matter of vital importance, viz., teaching and examining in the applied phases of the fundamentals.

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[1] Paper read before the National Confederation of State Examining and Licensing Boards, at Saratoga Springs, New York, June 9, 1902.

Henry Beates, Jr., in a paper entitled "How Should the Practice of Medicine Be Legally Defined?" calls attention to the lack of the proper legal definition of what constitutes the practice of medicine. This at present, he states, is as follows: "To practise medicine is to treat diseases and accidents by means of drugs or medicines, and that if the treatment of these is conducted without drugs and medicines, one so doing is not practising medicine!" This definition he believes to be insufficient, and insists upon the necessity of establishing a proper legal definition. He proposes the following definitions: **To practice medicine:** For any one, except those carrying out the directions of the attending physician, to engage, directly or indirectly, habitually or occasionally, gratuitously or for pecuniary or other compensation, in the care, management, or treatment, by any means whatsoever, either material or immaterial, for the relief or cure of any or all diseases, accidents, disabilities, to which human or animal life is exposed, threatened, or afflicted. **Practitioner of medicine:** Any one, except those carrying out the directions of the attending physician, who engaged directly or indirectly, habitually or occasionally, gratuitously, etc., as above. **The practice of medicine:** The engaging by any one except those carrying out the directions of the attending physician, directly or indirectly, habitually or occasionally, gratuitously, etc., as above."

The Illinois State Board of Health, on March 1, 1902, issued a summary of the laws **regulating and governing the practice of medicine in the United States.** The following tabulation gives a bird's-eye view of the whole subject. There are certain restrictions to the classifications which are indicated below.

The following States and Territories permit applicants to practise medicine on presentation of diplomas that are recognized by the Board of the State: Alaska, Arkansas, Colorado, Indiana, Indian Territory, Kansas, Kentucky, Michigan, Nebraska, Nevada, New Hampshire, New Mexico, Ohio, Oklahoma, Philippines, South Carolina, South Dakota, Virginia, Wisconsin, Wyoming.

The following States and Territories require an examination and the presentation of a diploma, with certain restrictions as indicated below: Alabama, Arizona, California, Connecticut, Delaware, District of Columbia, Florida, Georgia, Idaho, Illinois, Indian Territory, Indiana, Iowa, Louisiana, Maine, Maryland, Montana, Nevada, New Hampshire, New Jersey, New York, New Mexico, North Carolina, Ohio, Pennsylvania, Philippines, Puerto Rico, South Carolina, Virginia, Washington, Wisconsin.

The following States and Territories require examination, but do not insist upon diploma: Alabama, Hawaii, Indian Territory, Kansas, Massachusetts, Minnesota, Mississippi, Missouri, New Hampshire, North Dakota, Oregon, Philippines, Rhode Island, Tennessee, Texas, West Virginia.

The following States and Territories examine nongraduates in medicine: Alabama, Arkansas, Colorado, Hawaii, Indian Territory, Kansas, Massachusetts, Michigan, Minnesota, Mississippi, Missouri, Nevada, New Hampshire, New Mexico, North Dakota, Oklahoma, Oregon, Philippines, Rhode Island, Tennessee, Texas, Vermont, Virginia, West Virginia, Wyoming.

The following States accept a license of other States or countries in lieu of a diploma: California, Kansas, Maryland, Nevada, New Hampshire, North Carolina, Ohio, Pennsylvania, Virginia, Wisconsin.

Certificates of other Boards are recognized by the following States and Territories: California, Delaware, District of Columbia, Illinois, Indiana, Kansas, Maine, Michigan, New Hampshire, New Jersey, New York, Ohio, Pennsylvania, Puerto Rico, Texas, Virginia, Washington, Wisconsin.

Graduates of medical colleges within the State are licensed without examination by the following States: Illinois, Indiana, New Hampshire, Ohio, South Carolina, Tennessee, Wisconsin.

The following States and Territories permit physicians to practise in consultation in other States: California, Connecticut, Delaware, District of Columbia, Georgia, Idaho, Indiana, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Mississippi, Montana, New Hampshire, New Jersey, New York, North Carolina, North Dakota, Ohio, Pennsylvania, Philippines, Puerto Rico, Rhode Island, South Carolina, Tennessee, Texas, Utah, Virginia, Vermont, Wisconsin, Wyoming.

Restrictions.—In Alabama an applicant for examination before the County Board must present a diploma. When examined by the State Board, no diploma is demanded. In Alaska there is no law regulating the practice of medicine. Any physician by paying a license fee is entitled to practice. In Indiana an examination is required, except when the applicant has matriculated in an Indiana medical college before January 1, 1901, graduated and made immediate application to the Board prior to January 1, 1905. In the Indian Territory the Cherokee nation requires that a person desiring to practise medicine must show a certificate from a Board of Medical Examiners of the Superior Judicial Body. In the Creek nation graduates of a reputable medical college may practise without examination. In the Choctaw nation an examination is required. The laws are, however, actually null and void. In Kansas a physician is permitted to practise who furnishes a diploma from a college recognized by the Board. The Board may accept certificates of other Boards in lieu of an examination or diploma, at its own discretion. An examination is required if an applicant is not a graduate of a recognized medical college, and he must furnish evidence that he has attended a proper course of instruction. In Minnesota the Board of Medical Examiners will admit an applicant to examination without a diploma if he shows evidence of having attended 4 full courses at a medical college recognized by the Minnesota State Board. In Nevada the Board admits to examination persons who have practised medicine for at least 5 years in the State, and also graduates of foreign medical colleges. In New Hampshire physicians may practice without examination who have matriculated in a New Hampshire medical school before January 1, 1898, and who received their degree January 1, 1903. No examination is required of applicants examined and licensed by State examining boards in which the standard is not lower than that provided by the New Hampshire State Board. Applicants, if licensed to practise in some foreign country, or who have had 5 years' practice, are not required to show a diploma. In New Mexico the Board of Health will issue a certificate to a person who presents a diploma from a medical college in good standing; but if this college is not in good standing, an examination is required. In North Dakota the State Board requires an applicant to show evidence of having attended 3 courses of lectures of at least 3 months each. In Oklahoma an applicant must show that he has been engaged in the practice of medicine for not less than 5 years. In Ohio the State Board does not require an examination of a medical student who matriculated on January 1, 1900, in any medical college in the State of Ohio which is recognized by the State Medical Board and Registration of Ohio, and shall have graduated after January 1, 1900, and who shall have presented his diploma for registration prior to July 1, 1904. Physicians holding certificates issued by other State boards having equal regulations are not required to pass an examination. Applicants will be examined who present a diploma or license issued in some foreign country, if approved by the Board. In Pennsylvania the Medical Council will admit to examination an applicant who can show a license to practise medicine in some foreign country. Recognition of the license of another State is admitted provided that courtesy is reciprocated by that State. This is practically, however, inoperative. In the Philippine Islands certificates are granted without examination to persons who practised medicine in the Philippine Islands before the passage of an act regulating the practice of medicine and surgery in the Islands, December 4, 1901, who present a diploma from a reputable school or college, also to certain students in the St. Thomas University in the city of Manila, and to those who pass the examination before a board of examiners appointed by the United States Military Government of the Philippine Islands. In Rhode Island any reputable physician who passes a satisfactory examination is granted a certificate. South Carolina requires an examination when an applicant presents a diploma from "any college of established reputation in the State of South Carolina which has a 4 years' course of instruction, and a standard of not less than 75 % on examination, who gives satisfactory evidence of his standing to the State Board of Medical Examiners." In Tennessee the State Board does not require an examination from graduates of any reputable medical college in the State who shall have received certificates from the State Board of Medical Examiners prior to June 1, 1902. In Vermont "a person not a resident of the State, who has not received a diploma from a chartered medical college, shall obtain a certificate from a Board of Censors in the State, before he shall be permitted to practise the art of medicine therein." Virginia does not require an examination when the applicant shows satisfactory evidence of having graduated from some medical college chartered by the State or Territory in which it is situated, or when one holds a certificate from the examining board of any other State or Territory which shows that the applicant has passed a satisfactory examination, and has obtained a license to practise medicine from the State Board in the said State, Territory, or District. Applicants for examination must present a diploma if they are not eligible to a license without an examination. In Wisconsin the Board does not require an examination from a physician who holds a certificate from any other State Board imposing requirements equal to those established by the Board of Wisconsin. He must also present at the same time a diploma. This, however, is at the discretion of the Board. In Wyoming the Board examines applicants who may hold diplomas, certificates, or other credentials from institutions of other than recognized merit.

The Illinois State Board of Health, July 8, 1902, adopted a schedule of **minimum requirements for medical colleges,** to be considered in "good standing." Applicants must present a diploma or certificate of graduation from a high school, or present a certificate signed by the principal of a high school, or by the examiner of the faculty of a recognized college or university, or by the State superintendent of public instruction, or the superintendent of public schools, showing that an examination in the several branches taught during a 4 years' high school course has been satisfactorily passed. The faculty of a medical college has not the privilege of conducting a matriculate examination. On applying to the Illinois State Board of Health for a State certificate an applicant is required to show documentary evidence of a preliminary education, and must present also a medical diploma. Applicants may be allowed to enter in the second year of the 4 years' medical course, if they hold the degree of A.B. or B.S. or an equivalent degree from a college of arts and sciences in good standing. Applicants having a degree from a recognized college of dentistry or veterinary medicine may be admitted to advanced standing. Graduates of medical colleges may be admitted to any class without examination if the colleges have at any time been recognized by the Illinois State Board of Health. If the college is not recognized by the Board, no advanced standing whatever can be granted. The college course shall extend over 4 years, and shall consist of not less than 800 hours of work. The branches of medicine to be taught shall be at least as follows: (1) anatomy; (2) physiology; (3) chemistry; (4) materia medica and therapeutics (5) theory and practice of medicine, including ophthalmology, otology, dermatology, and neurology; (6) pathology and bacteriology; (7) surgery, including orthopedic surgery; (8) obstetrics; (9) gynecology; (10) hygiene; (11) medical jurisprudence (forensic medicine). The Illinois State Board of Health does not recognize any college as being in good standing the standard of which falls below the requirements as set forth in the schedule of minimum requirements, and which shall have not been in existence in good standing for less than 4 years, although the State Board has the privilege of recognizing a college which after personal investigation shall show that it complies with the schedule of minimum requirements. This schedule goes into effect on or after January 1, 1903.

MEDICAL ETHICS.

A correspondent wrote to the "British Medical Journal" for advice upon the following question of ethics: A patient had, while under the treatment of the correspondent, **consulted a specialist without telling her physician.** Later, however, she told the whole story, and stated that she had informed the specialist that she was under a doctor's care. He asked: "Is there one etiquette for general practitioners and another for specialists?" The question was submitted to a number of physicians and the conclusion was that a patient should inform his medical attendant before going to a specialist; that if a patient consults a specialist without informing the doctor under whose care he was at the same time, that the physician is justified in withdrawing from the case, although it may not be always advisable to do so. A specialist need not necessarily inquire if the patient is under the care of another physician, but should this knowledge come to him he should refuse to advise the patient without first gaining permission of the attendant physician. It is sufficient for the specialist to write to the attendant physician his opinion of the case, and the treatment advised. Finally, it is the duty of every physician to consent to his patient's seeking consultation.

Jervey[1] discusses the question of the **common law rights and the physician's prescription,** and concludes from a study of the medicolegal standpoint of this question that: (1) The patient has no legal

[1] N. Y. Med. Jour., July 20, 1901.

or other right to demand a written prescription or written directions from the physician. (2) It is right and wise that the druggist demand and procure from the physician his written orders for the compounding of prescriptions. (3) The physician has the undoubted right to designate what pharmacist shall fill his prescription. (4) The written prescription is simply an order from physician to pharmacist. It is, through courtesy, and by virtue of custom and convenience, handed to the patient for transmission; but the latter has not at any time the slightest right of possession in the instrument. (5) The druggist has at least the right of permanent guardianship (perhaps of outright possession) of the prescription, and he must keep it on file for reference and for any form of proper investigation. (6) There can be no right, extenuation, or excuse for a copy of a prescription, with physician's name attached, to be taken by druggist, patient, or any one else without the authority of the physician. (7) The careful physician should invariably retain a carbon-paper fac-simile copy of every prescription he writes. (8) The druggist has the legal right to utilize any formula that is uncopyrighted that may fall into his hands, but he cannot, unauthorized, use the name of its author in connection with it. In most States, however, statutes would bar his selling intoxicants or other poisons except by direct order of a physician. (9) If the druggist refills a prescription without the order of the physician who wrote it, he does so on his own responsibility, and he has no legal or moral right to leave or place the physician's name on the container. He proposes that physicians should patronize only "ethical druggists," by which he means those who confine themselves practically only to compounding physicians' prescriptions.

An editorial in the "New York Medical Journal,"[1] in discussing the **legal status of the unborn child,** takes up the question of the duty of the physician who is called upon to treat a woman in labor, where the complications are such as to make it probable that the child will not be born alive without measures which may prove fatal to the mother. This was a question raised by Zalackas,[2] and the novel point which he brings out in his article, as stated by the "New York Medical Journal," is as follows: "If we have no right to kill an unborn child to save the mother's life, we have no right to bring on premature labor in a case of contracted pelvis, for the reason that we have no more right to restrict a child's chances of survival even if we secure its birth alive, than we have to kill it outright." The editorial goes on to say that the "mere fact that some unusual obstacle is necessarily added to the child's chance for continued life does not, we believe, counterbalance that of the greater and more immediate danger to the mother's life involved in the cesarian operation." In the matter of the choice between the life of the mother and that of the child, Zalackas claims that the father has no voice in this matter, with which opinion the editorial agrees. Zalackas throws the responsibility of deciding this question upon the physician, in the event of the mother's refusing to undergo an operation the object of which is to save the child's life, which at the same time

[1] Aug. 17, 1901, p. 315.

[2] Progrès méd., June 29, 1901.

may cause the death of the mother. The "New York Medical Journal" does not agree with this opinion, although it believes that it is the duty of the physician to use all the arguments in his power to induce the mother to consent to an operation which he can assure her is attended with little risk to herself, and would result in almost absolute assurance that the life of the child would be saved.

The Comité consultatif de l'Assistance Publique[1] discussed the following question: **May physicians without violating professional secrets inform the authorities of evidence of crime** which the said physicians may receive through the exercise of their professional duties? They concluded that facts obtained through taking care of a patient should be considered absolute professional secrets.

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[1] Gaz. Hebdom. de Méd. et de Chir., April, 1902.

PUBLIC HYGIENE AND PREVENTIVE MEDICINE.

By SAMUEL W. ABBOTT, M.D.,

OF BOSTON, MASS.

MANAGEMENT AND CONTROL OF INFECTIOUS DISEASES.

Compulsory Notification of Tuberculosis.—After a full discussion of both sides of the question, a committee of the Society of Public Medicine, consisting of Vallin, Berthod and others, reported as follows[1]: "Notwithstanding the arguments which practising physicians propose in opposition to the practice of notification of cases of tuberculosis, and taking into serious consideration the burden imposed by a sanitary law making the attending physician responsible for neglect or refusal to report cases of tuberculosis, the Committee, while favoring the adoption of the principles, and not doubting for a moment that obligatory notification would prove an excellent and a desirable means in staying the spread of consumption, nevertheless hesitates to demand that this disease should be placed upon the list of contagious diseases. The Committee believes that the time has not yet arrived to adopt this course. They also believe that the desired result can be attained and the task of the physician relieved by educating the public as to the nature of the disease before placing it upon the list of unquestionably communicable diseases."

Use of Test Injections with Tuberculin in the Army.—Klinowitz[2] advocates the use of test injections with tuberculin in the army, since by such injections: (1) The appearance of latent tuberculosis may be prevented, and that of advanced tuberculosis may be arrested, among soldiers. (2) This method of preventing tuberculosis in the army may be carried out with less expense than under ordinary conditions.

Antituberculous Dispensaries in France.—Calmette[3] describes a dispensary which he has established at Lille, France, by the aid of public and private subscriptions to the amount of 76,000 francs (about $15,000). He also says that similar dispensaries have recently been established at Paris, Verviers, Huy, and Mons, and others are contemplated at Arras and at Nantes. He describes in detail the object of the work, its necessity, and the methods of operation. After collecting a subscription of 46,000 francs, the city gave the use of a lot of land and contributed a further sum of 30,000 francs for the undertaking. Very much depends, according to Calmette, upon the selection of a good assistant, who visits the homes of the sick and makes the necessary inquiries and gives proper instruction. He is provided with a blank form, which contains 68 questions as to the habits, necessities, and mode of life of the sick. In this instance the assistant is an old flax-carder, and secretary of the Mutual Aid Society. At the dispensary the usual clinical examinations are made, including the bacteriologic examination of sputum. Flasks are provided for the deposit of expectoration, together with a quantity of lysol as an antiseptic. An excellent circular is furnished containing instructions for the sick. Other subjects discussed in this paper are the cleansing and disinfection of the linen of the sick, the medical service of the dispensaries, the question of sending consumptives to the country to board, which the author discourages unless they can be placed where they may be sure of getting good food and attention, or the advantages of a sanatorium. The number of persons assisted at this dispensary from February 1, to June 1, 1901, was 236, of whom 157 were found to be tuberculous. The total expense for the four months was 5711 francs ($1142), or about $285 per month. This included the wages of the assistant, the cost of the pocket-flasks, disinfectants, etc. The medical services were gratuitous. For an account of a similar establishment at Liege by E. Malvoz, see also "La Presse Médicale."[1] The account shows that this dispensary has proved highly successful.

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[1] Rev. d'Hygiène, May, 1902, p. 460.

[2] Zeitschr. f. Hyg., etc., 40, 1, p. 154, 1902.

[3] Rev. d'Hygiène, vol. xxiii, July, 1901, p. 577.

A New Apparatus for the Sanitary Collection of Sputa.—Simonetta[2] describes an apparatus which, he believes, obviates the defects of previous appliances. It consists of a cone or a little horn of paper, 6 centimeters wide and 8.5 in height. They are very light, 10 weighing only 16 grams (½ oz.). They are easily made and fitted over a wooden cone, the edges being fastened with melted wax, which is also applied to the interior to make them impermeable and inflammable. A silken thread applied to each cone at the time of waxing serves for a handle to prevent soiling the hands. These may be collected in a box containing 120 cones. The box is lined with newspaper coated with wax and a layer of sawdust, and from this box the soiled cones are emptied into a tub or other receptacle to be taken to the furnace for cremation.

Transmission of Tuberculous Infection by Means of Cigars and Cigar-stumps.—Peserico[3] refers to this danger, and cites experiments in which tobacco saturated with the saliva of tuberculous patients was inoculated into guinea-pigs with the result of infecting 50 % of the animals. Other experiments made with cigar-ends collected from the streets resulted negatively, showing the rarity of Bacillus tuberculosis under such conditions, but not the impossibility of its existence.

The Aerial Dissemination of the Infection of Smallpox.—The well-known report of Power upon the influence of the Fulham smallpox

[1] March 2, 1901, p. 97.

[2] L'ingegnere igienista, Sept., 1901, p. 209.

[3] Sull 'importanza dei sigari e specialemente dei mozziconi di essi nella diffusione della tubercolosi. Annali d'igiene sperimentale, 1902, p. 149.

hospital in London, in spreading smallpox to the neighboring districts, followed as it was by similar instances in other parts of England, resulted in the transfer of all the smallpox hospitals of London to a number of ships moored in the river Thames at a point nearly 20 miles below the Tower Bridge. After the beginning of the present [1902] serious epidemic of smallpox in London (which is steadily on the increase), and the consequent transfer of many cases down the river to these anchored hospital ships, the disease broke out at Purfleet, a small district on the north bank of the Thames, about a mile from the ships. Thresh,[1] the medical officer of health of Essex, attributes the excessive prevalence of smallpox at Purfleet to the proximity of these hospitals of the Metropolitan Asylums Board of London. Cases followed one another in rapid succession since September, 1901, until about one-tenth of the population has been attacked, and it has spread to adjoining parishes. Vaccination has been much neglected in Purfleet: "The prevailing wind has been from the southwest, and in the cottages nearest, and exposed to the prevailing wind, out of every 8 persons, 1 has been attacked. If such an epidemic prevalence had occurred in London there would have been over a half-million cases in the past 7 months. My impression is that the infection may be carried 2 or possibly 3 miles in the direction of the wind." In an editorial in the same issue of the "Lancet," the same view receives substantial support in the following words: "These revelations are extremely inconvenient, and another result should be the recognition of the fact that we cannot, by isolation, hope to get rid of smallpox. The remedy is ready at hand, and consists in placing revaccination on the same basis as primary vaccination. With the German method we shall avoid smallpox outbreaks, but with primary vaccination alone we can never hope to do so."

On the other hand, in discussing this question, "Public Health"[2] reviews the reports of Power and Dr. Thresh. Before accepting Thresh's conclusion "Public Health" desires certain points to be cleared up; namely, the possible intercourse between the inhabitants on the shore with those on the ships, and whether there is any evidence that the crews or passengers of passing ships have contracted smallpox. It was also claimed that the 1200 boys and others upon training-ships stationed near the hospitals had not contracted the disease. This, however, is elsewhere explained by the fact that such boys are well protected by vaccination. In a full discussion of this subject in May, 1902, Scurfield doubts the conclusions expressed by Power, and cites the experience of the smallpox at Sunderland, England, in proof of his doubts. He states his "firm belief that smallpox has no more power of spreading through the air than any other infectious disease." Several other medical officers of health took part in the discussion, nearly all of whom expressed themselves as opposed to the views of Power.

Blanquinque[3] cites several epidemics of smallpox in different places in France in proof of the theory that its contagion is not carried through the air, but only by personal contact or by fomites.

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[1] Hospital Ships and the Dissemination of Smallpox, by J. C. Thresh, the Lancet, Feb. 22, 1902, p. 495.

[2] Sept., 1902, p. 683.

[3] Rev. d'Hygiène, July, 1902, p. 591.

Is a Smallpox Hospital a Nuisance?[1]—[Harrop and others *vs.* the mayor and corporation of Ossett, England.] The plaintiffs did not object to the use of the proposed hospital for other infectious diseases than smallpox, but that disease they alleged was liable to be spread by aerial convection. The hospital was only for 8 patients; only 1 had been so far received. It was 250 yards from a valuable mansion, 100 yards from the premises of a shoddy factory, about the same distance from the warehouse, and about 170 yards from a dwelling-house. A 6-foot wall, inclosing an open space of 40 feet, surrounded the hospital. *Held:* That the action must be dismissed with costs. Judge Romer, in giving his decision, said, *inter alia:* To sustain a *quia timet* action, the plaintiff must establish a strongly or extremely probable case that the apprehended mischief must arise. Here the evidence was most conflicting. If badly constructed, a smallpox hospital might be dangerous, especially if large and badly conducted, and the neighborhood crowded, but if well conducted, he was not satisfied that it would be such a source of danger as to constitute a legal nuisance. Considering the size, situation, and general character of the hospital in question, it would not be such a substantial danger as to justify him in granting an injunction. Moreover, there had been smallpox in the neighborhood, which had not caused ill results. It was not necessary to deal with danger which it was said would arise when the plaintiff's land was built on, until the danger arose. He thought there was no danger from drainage. Further, all the witnesses concurred that vaccination offered complete protection, and by taking this reasonable precaution all parties could protect themselves. Further, the case was not one of public, but of private, nuisance.

Smallpox in London in 1901.—Sykes[2] shows that one principal source of the spread of smallpox in London was the dirty linen sent to laundries. There was no evidence that laundries spread the disease to their customers, but received it from them, and that consequently those who handled the dirty linen were exposed to great risk. Another source was the existence of unrecognized cases coming from abroad and staying in the hotels and boarding-houses. By way of preventive measures he recommends the following: (1) International notification of smallpox; (2) extension of the instructions to be given to vaccination officials, as to suspicious rashes and ailments observed by them in the course of their duties; (3) the making of chickenpox a notifiable infectious disease; (4) the law should require that in every case of vaccination the operation should be thorough and efficient, and a description of the result obtained should form part of the certificate; (5) the provision of clinical examination in doubtful cases where bacteriologic examination is not available as an aid to diagnosis for the purpose of notification; (6) health authorities and the superintendents of smallpox hospitals should be in direct and continual communication with each other, and furnish each other immediately with any information necessary for the protection of the public; (7) a consideration of the powers and duties of health authorities with regard to the exclusion and isolation of persons who have been in contact with smallpox.

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[1] Hime's Practical Guide to the Public Health Acts, London, 1901, p. 21, Appendix.

[2] Smallpox in London, by J. F. J. Sykes, M.D., Medical Officer of Health of St. Pancras; London, Dec., 1901.

Smallpox.—The following data relative to the prevalence of smallpox, as determined by the mortality returns of different countries, have been compiled from such sources as are available, these being chiefly the annual statistical reports of the countries represented in the table:

DEATH-RATES FROM SMALLPOX PER MILLION INHABITANTS.

RELATIVE OR SIGNIFICANT FIGURES.

	German Empire.[1]	Scotland.[2]	Ireland.[3]	Sweden.[4]	Switzerland.[5]	Holland.[6]	England.	Italy.[8]	Japan.[9]	Belgium.[10]	Austria.[11]	British India.[12]	Hungary.[13]	Spain.[14]
1880	*	2.8	75.0	38.0	61.0	19.0	25.0	*	222.0	749	647	356	853	724
1881	*	5.1	14.0	65.0	59.0	18.0	119.0	*	351.0	487	819	362	787	624
1882	*	0.8	25.0	34.6	8.0	37.0	50.0	*	1122.0	277	962	430	765	1172
1883	*	3.0	3.2	27.2	9.0	160.0	36.0	*	327.0	314	605	1174	424	988
1884	*	3.7	0.2	12.6	22.0	15.0	83 0	*	11.0	234	523	1650	245	619
1885	*	10.0	0.8	0.9	149.0	7.0	104.0	*	88.0	280	575	450	287	*
1886	4.2	6.2	0.4	0.4	63.0	17.0	10.0	*	485.0	205	382	249	676	*
1887	3.5	2.1	2.8	1.1	5.0	4.0	18.0	550	156.0	102	417	313	932	*
1888	2.3	0.8	0.6	2.0	6.0	0 2	36 0	610	22.0	144	615	445	*	*
1889	4.1	2.0	——	0.4	1.0	2.0	0.8	449	8.0	199	515	570	*	481
1890	1.2	——	——	0.4	11.0	0.2	0.6	233	0.6	105	247	529	*	350
1891	1.0	——	1.5	0.4	9.0	2.0	1.7	96	18.0	212	285	426	*	506
1892	2.2	2.7	——	0.9	12.0	11.0	15.0	48	205 0	408	253	403	193	381
1893	3.1	16.5	2.0	5.2	5.0	40.0	49.0	86	289.0	334	243	274	69	265
1894	1.7	31.5	15.7	4.4	16.0	131.0	27.0	85	80.0	85	102	181	*	*
1895	0.5	11.5	31.8	——	0.3	16.0	7.0	97	6.0	47	47	188	*	*
1896	0.2	0.5	0.9	0.4	2.5	7.0	18.0	65	81.0	20	36	577	*	*
1897	0.1	2.4	0.6	0.2	0.3	0.2	0.8	32	*	21	57	696	*	*
1898	0.3	0 5	——	0.2	0.6	1.4	8.0	13	*	24	98	243	*	*
1899	0.5	0.2	0.2	0 2	*	——	5.0	7	*	38	73	211	*	*
Mean.	1.8	5.3	9.0	9.3	22.6	24.2	33.8	179	201.0	206	358	475	516	605

SOURCES OF INFORMATION FOR THE FOREGOING TABLE.

[1] Germany. Ergebnisse der amtlichen Pockentodesfallsstatistik im Deutschen Reiche (1886–1899).
[2] Scotland. Reports of the Registrar General of Scotland.
[3] Ireland. Reports of the Registrar General of Ireland.
[4] Sweden. Bidrag till Sveriges officiela Statistik, 1899, p. 40.
[5] Switzerland. Statistisches Jahrbuch der Schweiz, 1900.
[6] Holland. Bulletin de l'Institut International de Statistique, x, 83.
[7] England. Reports of the Registrar General.
[8] Italy. Statistica delle Cause di Morte, Rome, 1901, p. 12.
[9] Japan. Reports of the Central Sanitary Bureau, Tokio, 1900.
[10] Belgium. Annuaire Statistique de Belgique, 1901.
[11] Austria. Oesterreichisches Statist. Handbuch, Wien, 1900.
[12] British India. Annual Reports on the Sanitary Measures in India.
[13] Hungary. Bulletin de l'Institut International de Statistique, vol. x.
[14] Spain. Idem.

Vaccinal Establishments of Austria.[1]—Thirteen establishments were in operation in Austria in 1898 for the production of vaccine material. The quantities of such material issued were as follows:

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[1] Oesterreichische statistik. Sanitatswesens, 1898, Wien, 1901, p. xxi.

In 1893,	2953 points, 26,306 vials, and	enough in flasks to vaccinate	1,091,618	persons.
" 1894,		" " " " "	1,630,063	"
" 1895,		" " " " "	1,543,006	"
" 1896,		" " " " "	1,546,434	"
" 1897,		" " " " "	1,544,594	"
" 1898,	218,891 vials, and	" " " " "	1,254,990	"

Animal Vaccination in Italy.—Freyer[1] states that bovine lymph is exclusively used for vaccination in Italy. Retrovaccination of calves is not employed. Adult animals are mostly used, although young animals are usually employed when it is found necessary to rejuvenate a degenerated stock.

Dry Points and Glycerinated Lymph for Vaccinal Purposes.—Rosenau and others[2] conducted experiments to determine bacteriologically the comparative merits of dry points and glycerinated lymph, with the following results: Out of 41 dry points examined, there was an average of 4807 bacteria per point. Out of 51 glycerinated tubes and capsules, there was an average of 2865 bacteria per portion. This is an excess of what a good glycerinated lymph should contain. This difference in numbers does not justify the confidence placed in the glycerinated virus over the dry points as found in the market, judging from the limited number of counts made. So far as the kinds of organisms are concerned, pus cocci were found in both the dry points and the glycerinated lymph. "We think that we have demonstrated that some of the glycerinated lymph in the market is 'green;' that is, it is not kept a sufficient length of time before it is sold. From our studies we have concluded that we ought not to discredit glycerinated virus, for we consider its superiority amply demonstrated, but to condemn the practice of manufacturers who place an unripe product upon the market. Much of the vaccine sold must have a high initial contamination to contain an average of 2865 bacteria per tube, and it is evident that too great reliance is placed upon the glycerin."

Use of Waxed Bandages during Vaccination Period, in Animal Vaccination.—Pfuhl[3] gives the results of bacteriologic examination of lymph taken from calves at different dates after such taking, first, when tegmin bandages had not been used, and, second, when such bandages had been used. In the first instance (without bandage) the lymph 2 days after taking showed 836,000 germs per cubic centimeter; 39 days after taking, 1563 germs; 81 days after taking, 625 germs. In the second instance (with bandage) the lymph 2 days after taking showed 1350 germs per cubic centimeter; 39 days after taking, 238 germs; 81 days after taking, 75 germs. According to the experience of the vaccine establishments of the Empire, however, as detailed in the annual reports of the Imperial Health Board,[4] the advantages secured by the use of this bandage for protecting the vesicles upon vaccinated animals do not appear to have been in all cases satisfactory.

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[1] Vierteljahrsschrift f. gericht. Med. u. öff. Gesundheitswesen, 24, 4, 1902, p. 322.

[2] Amer. Med., April 19, 1902, p. 637.

[3] Viertelj. f. öff. Gesundheitspflege, Supplement, 1902, p. 180.

[4] Die Thätigkeit der im Deutschen Reiche errichteten staatlichen Anstalten zur Gewinnung von Thierlymphe während des Jahres, 1900.

The Sterilization of Vaccinating Instruments.—Sagrandi,[1] Surgeon of the 11th Dragoons (French), has invented and recommends for use in vaccinating large numbers of people, regiments, schools, etc., the use of vaccinostyles or small metallic lances, one for each person vaccinated. These are sterilized by placing them to the number of 50 or more in racks in a small metallic cylinder which is put into a large cylinder containing a 2 % solution of carbonate or borate of soda, which is kept at the boiling-point for 10 or 15 minutes by means of a spirit-lamp. The advantages of this method are greater rapidity in the vaccination of large numbers of persons, and the sterilization of the vaccinating instruments.

Insusceptibility to Vaccination.—It is quite probable that under the most favorable circumstances for vaccination (absolutely fresh lymph in each instance), insusceptibility in primary cases does not exist. Robertson says, in the twenty-fifth "Report of the Registrar General of Scotland:" "Constitutional insusceptibility, as expressed in the returns, is virtually a confession from an operator that he has made 3 unsuccessful attempts to vaccinate a child without ascertaining the cause of his failures, and the fact that a child has a certificate of insusceptibility does not absolve its guardians from the duty of having the reality of the conditions tested from time to time." Cory says[2] upon this point: "Of my own vaccinations, I may say that I have in my time performed over 38,000 primary operations with human or with calf lymph, and that it has only once fallen to my lot to fail twice at an attempt at vaccination. This subject was a 10-year-old child, in whom, as stated by its mother, vaccination had been attempted on previous occasions without result. My operation failed at the second attempt, and I did not get the opportunity to try a third time." Thorne,[3] chief medical officer of the Local Government Board, says: "I would point out that the number of consecutive primary vaccinations by the board's own officers, without the occurrence of a single instance of so-called insusceptibility, now reaches 107,180."

The Commercial Importance of Vaccination.[4]—Since the unfortunate modification of the Vaccination Law of England, enacted in 1898, very many children have been exempted from vaccination. The effect of this disastrous piece of legislation is already showing itself in more ways than one. Seventy English life insurance companies have required a renewal of their policies. Thirteen companies absolutely refuse to insure unvaccinated children, 57 companies consider such persons as exposed to an extra risk, and refuse to pay the amount insured if the policy-holder dies of smallpox, while only 10 companies continue the old form of contract. Many public departments refuse to employ unvaccinated persons. The Duke of Norfolk (postmaster-general), as a peer of the realm, voted for the conscience bill, but, as a matter of public policy, does not allow unvaccinated persons in the postal service.

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[1] Rev. d'Hygiène, Sept., 1902, p. 799.

[2] Lectures on Vaccination, p. 73.

[3] Twenty-seventh Annual Report of the Medical Officer of the Local Government Board, p. VIII.

[4] Rev. d'Hygiène, 1889, p. 1055.

The same is also true of the heads of the War and Navy Department, each of whom voted for the conscience bill, but allow no unvaccinated persons in their branches of public service. The first lord of the treasury has adopted the same measure. In some quarters of the city, landlords have turned out tenants who refuse to be vaccinated, and have placed signs upon their houses bearing the inscription "Not for unvaccinated tenants."

VACCINATION IN DIFFERENT COUNTRIES.

	German Empire.[1]	Scotland.[2]		Holland.[3]	England.[4]		Japan.[5]	Austria.[6]		British India.[7]	
	Vaccinations.	Successful Vaccinations.	Percentage of Births.		Successful Vaccinations of Children.	Percentage of Children Not Accounted For.	Vaccinations.	Vaccinations.	Percentage of the Living Born.	Successful Vaccinations.	Per 1000 of the Total Population Vaccinated.
1885	—	—	—	—	757,714	5.8	4,325,437	678,677	77.3	4,703,214	—
1886	—	110,864	86.6	—	754,059	6.4	4,551,235	691,460	80.3	4,950,893	—
1887	—	107,077	86.0	—	733,980	7.1	3,066,426	694,357	79.3	5,202,595	—
1888	2,187,484	106,734	86.6	—	719,103	8.5	2,295,988	727,802	82.0	5,640,967	—
1889	2,485,485	104,931	85.4	—	707,161	9.9	1,817,382	703,897	79.1	5,709,462	25.7
1890	2,463,268	103,635	85.2	112,594	682,560	11.3	1,937,959	710,034	79.0	5,974,598	28.9
1891	2,462,603	107,482	85.3	118,209	693,117	13.4	2,170,194	768,621	88.4	5,882,683	26.8
1892	2,144,821	107,352	85.8	128,756	663,657	14.9	3,988,761	816,767	88.8	6,396,480	27.5
1893	2,433,779	108,117	85.0	123,870	661,513	16.1	3,907,828	751,780	83.8	6,716,134	28.9
1894	2,534,040	106,840	85.8	163,412	626,126	19.2	3,022,541	749,708	81.2	6,869,271	30.3
1895	2,513,900	108,035	85.7	109,194	624,690	20.5	—	734,069	81.4	7,223,591	32.0
1896	2,533,227	110,033	85.1	117,653	602,922	22.9	—	819,592	87.1	7,309,087	32.2
1897	2,630,176	108,378	84.0	119,830	578,639	22.7	—	742,200	78.2	7,144,474	31.5
1898	2,676,015	108,981	83.2	124,257	562,737	21.5	—	734,989	77.8	6,860,915	30.2
1899	—	109,261	85.5	125,709	—	—	—	—	—	7,437,916	32.9

SOURCES OF INFORMATION FOR THE FOREGOING TABLE.

[1] Germany. Die Ergebnisse des Impfgeschäfts im Deutschen Reiche (1888–1898).
[2] Scotland. Reports of the Registrar General of Scotland (1886–1898).
[3] Holland. Reports of the Central Bureau of Statistics, 1900, p. 14.
[4] England. Reports of the Local Government Board (1885–1898).
[5] Japan. Reports of the Central Sanitary Bureau (1895–1896).
[6] Austria. Oesterreichisches Statist. Handbuch (1885–1900); also Statist. Sanitätswesen Wien, 1901.
[7] British India. Reports upon the Sanitary Measures in India (1885–1899).

Requirements as to Vaccination in the Public Schools of 19 cities.[1]—A physician's certificate of vaccination is required in the following cities: New York. (Rules and Regulations, Manhattan and the Bronx, 1900, p. 64.) Chicago, unless applicant has had smallpox. (Rules and Regulations, 1898, p. 30.) Philadelphia, unless applicant has had smallpox. (Rules, 1897, p. 292.) Boston, except when physician certifies that child is unfit for vaccination. (Rules and Regulations, 1900, p. 44.) This is also a State law and applies to every city and town in Massachusetts. Cleveland, vaccination or other protection. (Handbook, 1899-1900, p. 62.) Buffalo. (Charter and Ordinances, 1896, p. 231.) Detroit. (Rules, 1895, p. 24.) Milwaukee. (Rules and Regulations, 1891, p. 44.) Newark, unless child has had smallpox. (Report of 1899, p. 365.) Providence. (By-Laws, 1897, p. 20.) **Other Provisions:** St. Louis, children admitted, provided they have been vaccinated. (Rules, 1897, p. 78.) Baltimore, vaccination or other protection from smallpox required. (Rules, 1900, p. 15.) San Francisco, satisfactory evidence of vaccination required. (Rules, 1900, p. 38.) Cincinnati, satisfactory evidence of vaccination required. (Manual, 1895, p. 198.) New Orleans, satisfactory evidence of vaccination required. (Rules, 1896, p. 5.) Washington, vaccination or other protection from smallpox required. (Rules, 1900, p. 1.) Jersey City, vaccination or other protection from smallpox required. (Report, 1896, p. 148.) Louisville, evidence of vaccination or other protection required. (Manual, 1898, p. 23.) Minneapolis, evidence of vaccination, or physician's certificate that child should not be vaccinated, is required. (Annual Report, 1900, p. 134.)

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[1] Report of U. S. Commissioner of Education, 1899-1900, p. 2581.

Vaccination.—In the table on page 613 are brought together such facts relative to the extent of vaccination as were available from reports of different countries in the closing years of the last century. The figures represent exclusively public vaccinations performed by public officials at the public cost, and do not include such vaccinations as were performed by private physicians, and are consequently not contributable to the public records. It is very regretable that the relative figures in the table of vaccinations have so little relation to each other. This fact is due to the lack of uniformity in the methods of reporting such statistics in different countries.

Results of Antitoxin Treatment of Diphtheria.—The fatality of diphtheria in Chicago[1] from 1891 to 1895 was about 35 %. During the 63 consecutive months from October, 1895, to December, 1900, it was 6.79 %. A large proportion of the cases is treated on the first or second day of the disease. Of 5727 cases, 2 out of 476 were fatal when treated on the first day, or 0.4 %; and 1.5 % of them treated on the second day, 3.6 % of them treated on the third day, 11.4 % of them on the fourth day, and 23 % of them treated on the fifth day and later. In the report of the Massachusetts State Board of Health[2] for 1901 the following summary is given of cases which were reported by the Board from hospitals and from general practice. Out of 12,730 positive cases of diphtheria as determined by cultures taken, all of which were treated with antitoxin, there were 1329 deaths, or 10.4 %. Very many other cases were treated but no returns were made. The fatality, according to the day of administration, was as follows: Of those treated on the first day of illness, 7.8 %; second day, 6.1 %; third day, 9.6 %; fourth day, 13.0 %; fifth day, 15.6 %; sixth day, and later, 17.8 %.

Diphtheria Bacilli in Well Persons.—A committee of the Massachusetts Association of Boards of Health was appointed in 1900 to consider what was being done in American cities with well persons infected with diphtheria bacilli, and what were the views of health officers as to the proper methods of controlling such cases.[3] This committee has reported the following conclusions: (I) It is impracticable to isolate well persons infected with diphtheria bacilli, if such persons have not, so far as is known, been recently exposed to the disease. If it happens that such cases come to the knowledge of the health officer, it would, however, be wise to give instructions in regard to caring for the secretions, thus placing a part of the responsibility of the case upon the infected person himself. (II) It is not advisable as a matter of caution to isolate from the public all the well persons in infected families, schools, and institutions. Since the number of persons among the general public who are infected with diphtheria bacilli and who pass unrecognized and unrestrained may at any one time be greater than the number of infected persons in diphtheria families, it does not seem to be expedient to place restrictions upon the latter except when it can be done without causing much friction or hardship, or unless the danger in any particular instance can be shown to be very considerable. They recommend that children in infected families be kept away from day-schools, Sunday-schools, and all public places. Also that wage-earners may usually be allowed to continue their work; but that teachers, nurses, and others who are brought into close contact with children should not be allowed to do so. Milkmen should not be allowed to continue their business. In the bacteriologic portion of the report the committee states that the causal relation of the diphtheria bacillus to what is clinically known as diphtheria is based upon the following facts: (1) The Klebs-Löffler bacillus is the only species regularly present in diphtheria. It is most abundant at the height of the disease, and disappears soon after convalescence. (2) This bacillus produces a poisonous substance, or toxin, which after injection into animals produces inflammatory and hemorrhagic edemas, necrosis or sloughing of tissues, and paralysis. (3) The antitoxin produced by the injection of the toxin is specifically preventive and curative. For the purpose of forming conclusions, 3953 persons were examined, and the virulence of the bacilli was also tested in 86 cultures; and, in addition to them, 1536 other cultures were taken, the results of which could not be classified in consequence of the incompleteness of the records. It was found necessary to make allowance for the following factors: (1) The personal equation of the observer, under which must be included technic of preparation and classification. (2) The individuals from whom the specimens were obtained: whether they had been recently exposed to diphtheria or not; whether they were massed in such a way as to facilitate exchange of nose and throat contents. (3) Actual differences in locality, with the variables in conditions incident to this alone. While these investigations show that about 1 % to 2 % of all persons have typical Klebs-Löffler bacilli, only about 17 % of this 1 % to 2 % have virulent bacilli, or, in other words, 17 in 5000 to 10,000 of all persons have diphtheria bacilli which are dangerous to the public health. The number of virulence tests was too small to make these percentages of much value, yet they bring out the fact that if a healthy person is proved to have Klebs-Löffler bacilli, and there is no connection traceable between that person and cases of diphtheria, the chances are very much in favor of the bacilli being nonvirulent. Great care should therefore be taken not to put unnecessary restrictions on persons when the chances are great that the bacilli are nonvirulent. It is theoretically rational, if convalescents are isolated, to isolate also all healthy infected persons who have been exposed, until they are free from bacilli. Such a cause would undoubtedly result in preventing the occurrence of some cases, and in saving some lives. It is found, however, that such a course is not practicable for all bounds of health to follow. It is largely an economic question. The risk to the public health must be weighed against the loss and privation to the individual from isolation.

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[1] Monthly Bull. of Health Dept., Jan., 1901.

[2] Thirty-third Annual Report of State Board of Health of Massachusetts.

[3] Journal of Massachusetts Association of Boards of Health, July, 1902.

The Infectious Character of Typhoid Fever.—Borntrager[1] believes that too much attention is paid to the search for typhoid fever in the pollution of water-supplies, to the exclusion of other methods of infection.

Persistence or Survival of the Typhoid Germ in the Soil.—Rullmann[2] experimented with different soils, most of which had been sterilized, others not; all the cultures were exposed to the diffused light and to the temperature of an ordinary room. Under these conditions, with sterilized soil, there were few instances in which the germ was found virulent at the end of from 9 to 16 months. In the majority it could not be found at the end of a year.

Oysters and Typhoid Fever.—Sacquépée[3] cites an epidemic of typhoid fever at Rennes which occurred in 1901 and was traced to the use of oysters from a contaminated oyster-bed, and quotes the vote of the French Academy of Medicine, of June 30, 1896, to the effect that oysters taken from localities which are known to be contaminated should be exposed for 8 days before they are sold to the action of pure sea-water. He also suggests that laws are desirable which shall provide a guaranty of safety, so far as the location of oyster-beds is concerned.

Pathogenic Microbes upon the Surface of Plants which have been Sprinkled with Water Containing such Microbes.—Wurtz and Bourges[4] undertook certain experiments to determine whether it would be safe to eat raw vegetables which had been watered with water containing the bacilli of typhoid fever, tuberculosis, and anthrax. The plants were lettuce, radishes, and cresses. They found the typhoid bacillus in each instance, and the tubercle bacillus 18 times out of 30. Further experiments were made in which plants sprinkled with water containing different bacilli were exposed to the direct rays of the sun, showing the destructive action of such exposure. They also conducted experiments to determine whether germination of seeds and their subsequent growth would cause pathogenic germs to come to the surface of the ground after being buried in it. The results of these experiments, as the authors say, throw some light upon outbreaks of yellow fever and other diseases occurring where bodies of men and animals attacked with infectious diseases had been buried. They conclude that burial of such bodies in the ordinary manner is not sufficient, but that they should be burned or otherwise sterilized before burial.

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[1] Vierteljahrs. gerichtl. Medicin und öff. Sanitätswesen, 22, 1901.

[2] Centralbl. f. Bakt., 30, 1901.

[3] Rev. d'Hygiène, July, 1902, p. 577.

[4] Arch. de Med. Expérimentale, 1901, p. 575.

Milk-borne Epidemics of Scarlet Fever.—An account of an epidemic of scarlet fever believed to be due to the drinking of milk supplied from a dairy in Staffordshire, England, where several cases of illness resembling scarlet fever existed, is presented in "Public Health," December, 1901, page 128. Six London districts were invaded with the disease (Bethnal Green, Shoreditch, Finsbury, St. Pancras, Hackney, and Islington), in each of which the milk of this one dairy had been consumed by the sufferers. The account is compiled from the reports of Murphy, Bate, Bryett, and Newman, medical officers of health in these districts. An account of another epidemic of the same character appears in the same journal, page 138, by J. M. Martin, medical officer of health of Stroud, England. In this case a boy who helped to milk the cows was found to be ill on February 14, 1901. He had a "cold and sore throat," and several cases of scarlet fever occurred among the consumers of this milk in the following week.

The Fourth Disease.—Weaver[1] describes a disease differing essentially from the ordinary types of scarlet fever, measles, and German measles, for which reason several English physicians call it the "fourth disease." Dukes,[2] of Rugby, has alrsady described this as a scarlatinal form of German measles. The essential difference appears to be the form and character of the rash. That of German measles is described as "patchy," while that of the "fourth disease" is uniform, finely punctate, and indistinguishable from the ordinary early rash of scarlet fever. It appears first on the face, generally about the mouth. Secondly, even when the rash is extensive, the temperature is not high, not usually above 100° F., and the pulse is not accelerated. The throat symptoms are slight or absent, there is little or no feeling of illness and no loss of appetite. The incubation period is from 9 to 21 days. In none of the cases was there sneezing, coryza, or cough, and there was no enlargement of the cervical glands. There are said to be no sequels. Authorities differ as to the existence of desquamation. The practical importance of distinguishing these diseases consists in the possible admission of such cases to a scarlet-fever ward, where, as in one instance, the patient went through the usual course of "fourth disease" and then contracted scarlet fever and died of the latter disease.

Destruction of Rats as a Preventive Measure against the Plague.—Langlois and Loir[3] testify to the efficiency of the Clayton furnace for the destruction of rats and fleas in the holds of vessels from plague-infested ports. [This furnace, or one very similar in principle, has been in use for several years, for the disinfection of ships and cargoes at New Orleans.] Experiments conducted by the authors had demonstrated the great superiority of sulfurous acid gas over carbonic acid for the destruction of these animals. The Clayton furnace consists of a cylinder in which sulfur is burned while being fed with air by means of a fan or blower. From this the sulfurous acid is conducted by shafts into the room to be disinfected, or into the hold of a ship. When the disinfection is completed and the room or vessel has been kept clear for a sufficient time, the gas is drawn out, and dry, fresh air is supplied in its place by means of the same apparatus. By this method the percentage of gas obtained in a room or vessel is about 3 times as great as by the ordinary method. The rapidity with which the gas is evolved is also much greater than by the usual method. The authors conclude by saying that the "Clayton gas rapidly destroys rats and fleas, and in the case of ships it is not necessary to remove the cargo. It may, therefore, be conducted in the presence of the ship's company, and while the vessel is under way at sea." Experiments also showed that the effect of the gas upon the coloring of fabrics is trivial, and that most colors are not changed.

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[1] Public Health, Dec., 1901, pp. 143, 153.

[2] Lancet, July 14, 1901.

[3] Rev. d'Hygiène, May, 1902, p. 411.

The Prevention among Workmen of Anthrax and Other Diseases Transmitted by Means of Rags, Raw Hides, Hair, and Bristles.—Gruber[1] treats of those industries in which the workmen are especially liable to the infection of anthrax and other diseases. The dust of rags is peculiarly liable to communicate smallpox, and special means should be provided for ventilating the rooms in which rags are sorted and special tables should be provided upon which the work may be conducted. [These measures are successfully carried out in the Cumberland Paper Mills, near Portland, Maine.] He believes that steam is the only method by which rags can be successfully disinfected, and the cost of its application makes the work prohibitive. [The impossibility of successfully disinfecting baled rags by means of steam by any method hitherto devised was demonstrated by attempts made at Boston in 1887.] For dried and salted hides and skins, practicable methods of disinfection which are satisfactory to the trade do not appear to have been introduced. The author recommends for preventing infection from hides, a certificate of their source; the establishment of tanneries at a distance from habitation, provided with the best sanitary conditions; information supplied to workmen, showing them how to detect the first appearance of malignant pustule; protection of the hands by means of gloves; frequent antiseptic baths in the course of their work; and precautions in the unpacking of the dried skins. For horsehair and pigs' bristles he recommends steam under pressure for ½ hour, or immersion in boiling water for several hours. The application of potassium permanganate in a 20 % solution, followed by decoloration with sulfurous acid, or the use of formaldehyd, has given encouraging results.

The Prevention of Leprosy.—Gerber[2] recommends the following measures for the prevention of leprosy: (1) Special attention should be given to leprous affections of the upper air-passages, and the exposed parts should be protected as much as possible by local treatment. (2)

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[1] Das Österreichische Sanitätswesen, Dec. 5, 1901.

[2] Hyg. Rundschau, Sept. 15, 1902, p. 938.

The most important vehicles of infection are the secretions of the nose and throat, which should be destroyed. In leprosy the nasal secretion plays much the same rôle as the sputum in cases of pulmonary tuberculosis. (3) As an indirect vehicle of contagion, the linen should also receive special attention, especially handkerchiefs. It is better to use clothes which can be destroyed immediately after use.

Destruction of Mosquitos as a Means of Preventing Disease.—Ross[1] summarizes the work of mosquito destruction as follows, speaking from his own experience and that of others: **Objects**: (1) We do not propose to exterminate mosquitos in any entire continent. We propose only to deal with them in the town in which we live, and in its suburbs. (2) We do not propose to get rid of every mosquito, even in this town. We aim only at reducing the number of the insects as much as possible. (3) We do not think it possible to drain or otherwise treat every breeding-place in the town. We aim at dealing with as many as possible. (4) We cannot exclude mosquitos which may possibly be blown into the town from miles away. We content ourselves with preventing the insects from breeding in the town itself. **Methods**: (1) We start work at once with whatever means we can scrape together. (2) We operate from a center outwards. (3) We clear houses, back-yards, and gardens of all rubbish; empty tubs and cisterns containing larvæ, or destroy the larvæ in them by means of oil. (4) We show people how to do these things for themselves, and how to protect tubs and cisterns by means of wire gauze. (5) When we have cleared as many houses as we determine to deal with, we clear them over and over again. (6) We fill up or drain away all the pools, ditches, old wells, and puddles we can, especially those which contain most larvæ. (7) Such pools as cannot be filled up or drained are deepened, and cleared of weeds, if they contain larvæ. (8) Streams and water-courses which possess larvæ are "trained." (9) When we can do nothing else, we destroy the larvæ periodically with oil, or by brushing them out with brooms, or by other means. (10) We endeavor to interest our neighbors in the work, and to educate the town into maintaining a special gang of men for the purpose of keeping the streets and gardens absolutely free of stagnant, mosquito-bearing water. Finally, the first law of tropical sanitation should be, "**no stagnant water.**"

Paludism without Malaria.—Celli and Gasparini[2] cite instances in which all the known requisite conditions for malaria exist, without the appearance of the disease. They have made observations in the marshy regions of the coast and interior, southward from Leghorn. These regions, during the past 25 years, have been comparatively exempt from malarial fever, notwithstanding the existence of the marshes, canals of stagnant water, rice plantations, bogs, and ditches used for the soaking of hemp. These stagnant pools of water contain the larvæ of Anopheles, of the varieties claviger and pictus, in abundance. They also find persons who have contracted malaria elsewhere, but, notwithstanding the existence of these two factors, Anopheles and certain malarial subjects, epidemics of the disease do not occur. Children brought up in these marshy places have the appearance of perfect health. Those peasants who have an unhealthy appearance owe their condition to pellagra, but not to malaria, and although they work at night they do not contract the fever. This entire marshy territory has only two small and very circumscribed foci where malaria exists. Quinin is used neither more nor less than in other places. Here, then, we have all the recognized conditions for epidemic outbreaks, stagnant water, abundance of Anopheles, and persons who have contracted malaria elsewhere, but no spread of the disease. These circumstances appear to be unexplainable.

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[1] Mosquito Brigades and How to Organize Them, 1902, p. 42.
[2] Il Policlinico sezione pratica, 1901.

Trichinæ in Dogs.—Out of 243 dogs killed or examined at Chemnitz[1] in 1899, 3, or 1.23 %, were found infected with trichinæ.

Trichinosis in Prussia.—The official examinations[2] of swine for trichinosis in Prussia in 1899 embraced the inspection of 9,230,353 hogs, among which 1021 were found affected with trichinosis, or 0.11 %. Of these, the greatest number (444) were found in the district of Posen.

Rabies in Germany.—The number of animals killed in Germany in 1899 on account of rabies was less than that of the previous year; they were as follows: 911 dogs, 7 cats, 9 horses, 171 cattle, 38 sheep, 1 goat, 17 swine—in all, 1154 animals. In addition to the foregoing 1220 vagrant dogs and 2564 others suspected of infection were killed. In other countries in the same year there were 299 outbreaks in Belgium, 10 in Great Britain, 47 in Italy, 180 in Roumania, 140 in Bosnia and Herzegovina, 2331 in France, 268 in Algiers, and 2 in Switzerland. The incubation period as observed varied among dogs from 4 to 133 days, among cattle from 8 to 480 days, and among swine from 12 to 30 days.

The Methods of Conveyance of Yellow Fever Infection.—Carter,[4] in a logical argument upon the methods of yellow fever infection, concludes that this disease is conveyed in nature only by a host and doubtless by a mosquito host. Since (1) there is the analogy of other diseases conveyed by insect hosts; (2) that all facts observed about the propagation of yellow fever agree with the necessary deductions of this theory; (3) no other theory explains all the facts observed of its propagation.

Old Wall-papers and the Danger of Infection. The "Sanitary Record"[5] calls attention to two customs liable to spread infection; (1) The repapering of tenement-houses by covering the old paper instead of stripping it off; (2) whitewashing walls without first cleansing them by washing. In a discussion upon this subject, one of the speakers related an instance "in his own experience where 17 layers of paper had been taken from a wall, and there was no more fruitful source of infection, especially to painters, than the removal of old paper, which might have covered up infection." The custom of putting on several layers of paper one over another was characterized as a "vicious habit."

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[1] Ostertag's Zeitschr., 1900, p. 117.
[2] Jahresber. über d. Verbr. der Thierseuchen, 1899, 14, 98.
[3] Jahresber. v. Thierseuchen, 1899, 14, 24.
[4] Bull. No. 10, Yellow Fever Institute, Washington, D. C., 1902.
[5] March 28, 1901, p. 267.

Infection by Kissing.—Giovanni[1] calls attention to the perils of transference of disease by kissing relics and statues, rings, crucifixes, and images of monks and nuns. These are common Italian practices at festivals and pilgrimages, and are often performed by persons in feeble health, or who have just recovered from dangerous diseases. Casagrandi examined certain statues which had often been kissed, and found upon them the specific bacteria of tubercle and diphtheria, besides other pathogenic microorganisms.

Reception-houses for Persons Who Have Been Exposed to Infectious Disease.—Under the provisions of the Public Health Act of Scotland, houses may be provided for the reception of persons who have been exposed to infection. In commenting upon the practice, the Local Government Board of Scotland says:[2] "In our opinion, reception-houses should be entirely dissociated from hospitals for infectious diseases. Such hospitals are properly suburban, whereas houses of reception ought to be urban, convenient to the ordinary residences of the persons who may most probably be required to use them. They are not prisons, but places substituted for ordinary residences, from which inmates may go in and out so long as they are in health, and their persons and clothing free from infection. The only difference, or the essential difference, between life in a reception-house and at home is that in the former case the inmates live under skilled observation. We pointed out in some cases that the place submitted partook too much of the nature of barracks. There was no suggestion of home about them, and no provision for the accommodation of families. There was no other division of inmates than according to sex, and subject to that division they were thrown together. There were no rooms where parents and young children might sleep, or any possibility of separating the respectable artisan and his adult sons and daughters from the tramp or person of doubtful character. The best reception-houses are adapted from large self-contained houses, and if it be necessary to erect a building on purpose, this is the idea to be kept in view."

Prevention of Ophthalmia in the Newborn.—Pinard[3] presented a report upon this subject to the Academy of Medicine, referring to the necessity of immediate treatment in cases of ophthalmia neonatorum, and recommending certain well-known remedies: The Academy acted favorably upon the following conclusions: (1) A brief circular should be distributed in all the cities, indicating the causes, symptoms, and dangers of ophthalmia in newborn children. (2) Measures should be taken for securing the notification of every case. (3) Oculists should be employed by every lying-in establishment who shall direct the curative treatment of such cases and instruct the attendants and midwives.

The Increase of Cancer.—Geirsvold[4] alludes to the voluminous literature upon this subject, consisting of 5500 articles in the past 15 years and from which some valuable conclusions may be drawn. The

[1] Rivista Medica, July, 1902.
[2] Annual Report for 1900.
[3] Bull. de l'Acad. de Méd., July 30, 1901
[4] Kringsjaa, October, 1901, Christiania.

geography of the subject indicates a preponderance of cancer in the temperate zones and among civilized nations. It appears to be a disease attendant upon civilization, following the migrations of European nations into their colonies. In Norway, from 1878 to 1897, four-fifths of the cases of cancer were those of the organs of digestion, including the stomach, the alimentary canal and its appendages, the mouth, the tongue, and the pharynx. Cancer is attributed to all sorts of causes by different writers, including food, water, soil, occupation, contagion, etc. In another paper Geirsvold[1] states that cancer has increased in Norway from a mortality of 1.9 per 10,000 in 1865 to 4.2 in 1880 and 8.5 in 1897. In the western part of Norway, and especially among the valleys and fiords, cancer is rare, and although regions of extreme heat and cold are comparatively free from cancer, there appear to be centers of the disease in the Loffoden Islands in latitude 68°, and the extreme northern districts of Tromso, Vardo, and Hammerfest (lat. 69° to 71°), these latter having a cancer death-rate of 11, 8.8, and 13.5 per 10,000 inhabitants, the latter being the highest in the kingdom.

DISINFECTION.

Disinfection of Books.—The syndicate of Hungarian libraries requested the National Society of Hygiene to publish a resolution forbidding the sale of second-hand school-books. Krausz[2] undertook a series of experiments to determine the question of danger. He soiled the leaves of books with cultures of different pathogenic germs. The cholera germ lost its vitality when thus exposed in less than 48 hours, the diphtheria bacillus in 28 days, the staphylococcus in 31 days, that of typhoid fever survived 40 or 50 days and once to 95 days. That of tuberculosis remained doubtful after 103 days. He found it possible to disinfect books by means of steam without serious injury. The pages were not changed, but the covers were slightly injured. The time of exposure to steam was 40 minutes. He does not think it necessary to disinfect the books of classes which are not changed during the school-year, provided the change is not made till after the close of the long vacation.

Experiments with "Lysoform," a New Disinfectant.—Symanski[3] states that, owing to its very slightly poisonous nature and to its powerful disinfecting properties, lysol takes high rank among the more recent preparations placed on the market, and if only for this reason this new substance discovered by Stephan and produced from formalin, which has been known in its pure state as "lysoform," merits attention as an industrial disinfectant. It possesses very marked disinfecting properties, it is not irritating to the skin, it is nearly devoid of smell, and it is nonpoisonous. It is in appearance a clear yellowish fluid of an oily consistency, which mixes readily with water or alcohol, and when shaken up produces a copious froth very similar to soapsuds. The author describes the various tests undertaken to examine the relative action of lysol and lysoform in dilute solutions, and shows that the latter possesses very decided antiseptic properties. He also carefully tested its action upon small animals to demonstrate the absence of toxic action. As a disinfectant it matters but little whether it is used hot or cold. If employed as a 3 % solution, it forms an excellent wash for the hands and is wholly without action on the skin.

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[1] Nordiskt Medicinskt Arkiv, 1901, II, 2, R. 11.

[2] Zeitschr. f. Hyg. u. Infections-krankheit., 38, 1901.

[3] Zeitschr. f. Hyg., vol. XXXVII, pt. 3, p. 393.

Javelle water (chlorinated soda sol.), having become somewhat neglected as a disinfectant, Bezançon[1] recommended its use, and cited experiments in which it was used successfully for the disinfection of the sputa of consumptives. Vallin and Martin cited similar experiments, but found that it was more or less injurious to fabrics. They recommended a solution in water of 1 : 30 parts or 1 : 50. When used with linen or clothing, articles should be thoroughly rinsed afterward to remove all traces of the chlorin.

Mercurial Disinfectants.—Bormans[2] reports the result of certain experiments with several preparations of mercury (cyanid and potassic iodid) as disinfectants, and comes to the conclusion that neither of these salts are as valuable as mercuric chlorid. He states that he has yet to find a disinfectant which has the germicidal power while also lacking the poisonous quality and the practical defects of the mercuric chlorid.

FOOD AND DRUG INSPECTION.

Substitutes for Bread in Time of Famine.—Erismann,[3] after experimenting with various substitutes for wheat-bread, such as rye flour, oatmeal, barley, maize, buckwheat, acorns, straw, sunflower seeds, and other products, does not recommend their use in time of famine as additions or mixtures for use in making bread.

Boric Acid as a Food-preservative.—Kister[4] experimented with boric acid as follows: Three healthy persons were given daily 3 grams of boric acid spread upon bread and butter. All 3 were attacked with loss of appetite, vomiting, and diarrhea. Two had albuminuria, which disappeared after they had ceased taking the boric acid. This dose of boric acid, as the author says, might easily be taken daily by persons who eat articles of food preserved in this manner. [This is doubtful.] Out of 6 persons who took 1 gram a day, 4 had disturbances of the digestive organs. He concludes after further experiments on animals that the use of this preservative should be forbidden.

Borax or Boric Acid as Food-preservatives.—Vaughan and Veenboer[5] have reviewed the recent literature of borax and boric acid as food-preservatives, and present the results of certain experiments which they had conducted, in which chopped meat, butter, and cream were treated with definite quantities of borax and boric acid. They conclude as a result of these experiments that the amount of boric acid to be allowed in chopped meats should be limited to 0.5 %, but that there was no objection to the use of as much as 1.5 % when it is to be sprinkled on the surface of the meat, since most of this is washed off again. They also give the following conclusions, both from their experiments and from the literature already published: (1) The use of borax or boric acid as a preservative in butter and cream in the quantities specified in the recommendations of the English Commission is justified both by practical results and by scientific experimentation. (2) The dusting of the surfaces of hams and bacon which are to be transported long distances with borax or boric acid, not exceeding 1.5 % of the weight of the meat, is effective and not objectionable from a sanitary standpoint. (3). Meat thus dusted with borax or boric acid does not become slimy because the preservative thus used prevents the growth of aerobic, peptonizing microorganisms.

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[1] Rev. d'Hygiène, Dec., 1901, p. 1081.
[2] Rivista d'igiene e Sanità pubblica, Oct. 16, 1901, p. 778.
[3] Zeitschr. f. Biol., 42, 1901, p. 672.
[4] Zeitschr. f. Hyg. u. Infections-krankheit. 37, p. 225, 1901.
[5] Amer. Med., Mar. 15, 1902, p. 421.

The Detection of Arsenic in Food and Drink.—Thompson[1] lays down the following requirements for the detection of arsenic in articles of food and in beverages: (1) The results are obtained quickly; (2) the personal equation should, so far as possible, be eliminated, so that two chemists would obtain, as nearly as possible the same result when working on the same sample; (3) as it yet remains uncertain how much arsenic may be taken without injury to health, cognizance should be taken of very minute quantities of arsenic up to, say, the $\frac{1}{1000}$ of a grain of arsenic trioxid per pound avoirdupois of solid, or per gallon of liquid.

Influence of Alcoholic Liquors upon Public Health in Russia.—Sikorsky[2] states that 84,217 persons died of acute alcoholism in Russia during the years 1870–1887, or 4678 per year. He gives to alcohol the name of the "great assassin." He states that the death-rate from acute alcoholism in France is only 11.5 per million inhabitants, in Germany 12, but in Russia it is 55.2 per million. He finds that acute alcoholism is associated with low temperatures, and claims that he has established the fact that extreme cold acts like an added dose of alcohol. In northern Russia, where the consumption of alcohol is least, the deaths from acute alcoholism are 96 per million, as compared with 18 per million in southern Russia.

The Milk Supply of London.—A writer in the "British Food Journal," September, 1901, estimates that the "total quantity of milk required to meet the wants of London during 12 months reaches the enormous total of 51,840,000 gallons, or about 142,028 gallons per day; more in summer, less in winter,—in the poorer districts of London doling out the precious liquid food in such small quantities that the smallest coin of the realm, as regards its face value, has to be employed in payment for the same. The same writer estimates the amount necessary to supply the yearly total for the United Kingdom at 473,652,980 gallons.

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[1] Brit. Food Jour., Aug., 1902, p. 170.

[2] Influence des boissons alcooliques sur la santé et la morale publique en Russia, par le Prof. Sikorsky, Kiev.

New Milk Standard in England.—The Department of Agriculture has established a new standard of milk,[1] which went into operation September 1st. This regulation makes 3 % the standard of milk-fat, and 8.5 % for the solids other than fat.

The Use of Food-preservatives.—Hagemann[2] publishes the following conclusions relative to the use of food-preservatives: (1) Any chemical mode of preserving milk is to be condemned. (2) A radical abandonment of the use of preservatives in alcoholic liquors is not yet practicable. In wines the sulfites are required, and beer must be treated with salicylic acid for export. (3) For preserving the juices of fruits, chemical preservatives are unnecessary. (4) The methods of chemically disinfecting drinking-water, so far as they have been made known, are valuable acquisitions.

SCHOOL HYGIENE.

The Orientation of the School-house.—Bürgerstein[3] gives the preference to south and east exposures for the principal school-rooms. A northeast is better than a northern exposure. Such a room will receive some direct sunlight, and there will be but little difficulty in the management of curtains. A building facing the southeast has the advantage of having three of its sides exposed to the sun through the greater part of the year.

School Seats and Benches.—Janke[4] gives the following definitions of the two most important measurements having relation to school seats and desks. The **difference** is the vertical distance from the inner edge of the desk to the seat, or to the extended edge of the seat. This distance must be such that the edge of the desk shall be about opposite the navel, so that the pupil, when sitting erect, may place the arm upon the desk to write with the elbow bent a little at one side and in front of the pupil. The **distance** is the horizontal interval between the front edge of the seat and a vertical line let fall from the edge of the desk. If this line coincides with the edge of the seat, the distance is called a zero distance; if the line falls in front of the edge of the seat it is a plus distance; if behind it, a minus distance.

Movable School Seats and Desks.—Eulenberg[5] quotes Barnard as the first in the United States to establish movable desks and seats; he also quotes Prausek, of Vienna, as stating the following advantages of movable desks and seats: (1) Each scholar can be furnished with a seat adjusted to his individual needs. (2) The seat may be moved and thus conveniently adapted to writing, drawing, or other school exercises. (3) In the school-room the floors may be cleaned more easily

[1] Sanitary Record, Sept. 12, 1901, p. 232.

[2] Vierteljahrs. gericht. Med. u. Off. Gesundheitswesen, April, 1902, p. 373.

[3] Handbuch der Schulhygiene, p. 11, 1901.

[4] Grundriss der Schulhygiene, 1901, p. 111.

[5] Schulgesundheitslehre, vol. I, p. 271, 1900.

and thoroughly than with fixed seats. (4) The cost is less. (5) By the use of movable desks and seats the provision of separate rooms for drawing, and for sewing or other work for girls, may be dispensed with. (6) The use of such seats prevents overcrowding and provides a comfortable seat for each scholar. (7) School tables can be used at each of which 4 or 5 scholars can sit. (8) Such seats and desks allow freer use of class-rooms for gymnastic exercises.

Sanitary Education of Children.—Chapin[1] urges the importance of special teaching in the matter of cleanliness among children. One reason of the spread of infectious diseases is the existence in the community of a considerable number of infected persons who are only very slightly ill, or not ill at all. It is these unknown sources of infection which cause much trouble. In order to diminish them it is desirable to teach the people, especially the children, the best methods of protecting themselves against infection, and since the infection of several diseases is contained in the secretions of the nose and mouth, much good can be accomplished by means of education in this direction. In the language of the writer, "children should be taught that their bodies are their own private possessions, that personal cleanliness is a duty, that the mouth is for eating and speaking and should not be used as a pocket, and the lips should not take the place of the fingers."

School-room Floors.—Bernstein[2] publishes the following recommendations regarding school-room floors: (1) The character of the floor has a direct bearing upon the cleanliness of the school-room. The rougher and more cracked it is, the more difficult and expensive to keep it clean. A covering of linoleum is best. (2) Seats fastened to the floor make cleaning difficult. (3) Moist sawdust may be used with advantage in cleaning floors of school-rooms. (4) If the floors have tight joints, washing may be recommended. (5) The expense of cleaning may be considerably diminished by the provision of a good floor and proper seats and desks at the outset. (6) A bad floor should be repaired by planing and filling the cracks. This operation should be repeated once a year. The author recommends the use of oil. [Oil is not so good as some of the newer solutions of wax now in use, since these do not darken the floors so much as oil.]

Linoleum as a Sanitary Covering for Floors.-Biancoth[3] reports the results of experiments to determine the qualities of linoleum. He found its power of conducting heat was low and almost the same as that of wooden floors, its permeability to water is also very slight, it also resists the application of most disinfectant solutions except a 10 % solution of carbolic acid, and pathogenic germs find no lodging-place in it, as they do in the cracks of floors, either of wood, brick, or stone.

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[1] Med. News, Mar. 8, 1902.

[2] Vierteljahrs. f. öff. Gesundheitspflege, 3, 1902, p. 510.

[3] L'ingegnere igienista, 1, 1902.

WATER-SUPPLY AND SEWAGE.

The Hunermann Process for Disinfecting Water.—Schuder[1] in a recent paper states that under Hunermann's system a 10 % solution of sodium chlorid is employed, and in all cases determinations were made first of the contents in chlorin before use. To remove the superfluous chlorin, a solution of sodium sulfite prepared by the author was invariably used. The precautions taken to carry out the various experiments are set forth. Four different kinds of water, representing the chief varieties of hardness, impurity, etc., were selected for the tests: viz., distilled water, spring water, service-water, and canal-water. To each of these samples, mixed with the chlorid, test-cultures of cholera vibrios and the bacilli of typhoid fever and diarrhea were added in stated proportions, and the results were compared with the untreated samples of the same kinds of water. The various sets of tests are arranged in a tabulated form, and Schuder states that the process appears to reduce to a very marked extent the vitality of the germs, even in highly polluted samples of water; in some instances the water was rendered germ-free. The cholera vibrios were in a few of the tests wholly destroyed; but neither in the case of cholera or typhoid fever germs was the treatment entirely reliable. Even the bacilli of diarrhea, which are much more susceptible than most other pathogenic germs, were not killed in all cases. Hunermann's process yielded better results, on the whole, than the bromin process of Schumberg. The author appends a series of conclusions bearing on the merits of plate cultures and other matters needing special attention.

Sewage Purification and Water Pollution in the United States.—In an article in the "Engineering News,"[2] the present extent of sewage purification in the United States is partly shown by a table of 95 cities and towns, with a population of 3000 and upward. About 9 years ago only 31 cities and towns in the United States had sewage purification works, according to a list then published. Each list contains a number of Western towns where all the sewage is utilized for irrigation purposes, and not treated from sanitary motives. The table gives the method used for the disposal of the sewage. The method of sewage purification principally in favor at first was intermittent filtration. Table II, which is a classified summary of Table I, shows that this method still takes the lead, numbering 24 cities and towns against 9 for broad irrigation and sewage farming, and 7 each for septic tanks and chemical precipitation. Besides this, in some places septic tanks are combined with other methods of purification. Chemical precipitation never made much headway in the United States, and at present it is almost at a standstill. The septic tank is comparable with chemical precipitation, but is more popular, because it entails no expense of chemicals, and, it is claimed, comparatively little labor or expense for sludge disposal. Contact and bacteria beds cannot yet be spoken of with much confidence. A careful examination of the whole list of 95 places indicated that, say, 60 places in the United States, with a population of 3000 and upward, have really creditable sewage-purification plants; but the number is increasing rapidly. Table III gives a list of lawsuits for water pollution which shows that this increase is inevitable.

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[1] Zeitschr. f. Hyg., 1902, p. 379.

[2] April 3, 1902, p. 275.

Sanitary Condition of Hamburg before and after the Introduction of Filters.[1]—

Years.	General Death-rate per 1000.	Typhoid Death-rate per 100,000.	Total Deaths from Typhoid Fever.		
				City.	Suburbs.
1888	25.3	54.3	295		
1889	23.9	42.8	243		
1890	22.3	27.0	160		
1891	23.6	23.6	151		
1892 (Cholera year.)	39.8	38.7	216		
Filters introduced about the middle of 1893.					
1893	20.4	18.1	115		
1894	17.9	7.1	47	37	10
1895	18.9	10.7	72	57	15
1896	17.1	5.6	39	35	4
1897	16.9	7.7	55	47	8
1898	17.3	4.5	33	31	2
1899	17.3	4.2	31	26	5

Experiments in Sewage Disposal.—Frye[2] concludes as the result of certain experiments with filtered sewage sterilized by mercuric chlorid, and by exposure to steam at 100° C., that the sterile filters did about one-third of the work done by the normal filters. The oxidation of the sterile sewage as measured by the nitrites and nitrates was one-seventeenth of the oxidation which took place in the normal sewage. He therefore concludes that the oxidation of sewage containing no bacteria is very slow, or, in other words, that the chemical oxidation due to the oxygen in the air is unappreciable. In an appendix in the same report Houston discusses the bacteriologic qualities of crude sewage and sewage effluents, and the question of standards in relation to potable and nonpotable streams. With reference to potable water, he says it would be reasonable to require that an effluent be free, or nearly so, from putrescible matter, as judged by chemical standards, and be free also from the specific germs of epidemic disease; *e. g.*, B. typhosus as judged indirectly but seemingly in safe fashion by the absence of B. coli. It might be convenient to fix some limit; *e. g.*, that B. coli must be absent from 1 cc. of a sewage effluent; that if this end could be attained by filtration or other method, not to consider the use of chemical substances essential. With reference to nonpotable streams, he suggests that the discharge of an impure effluent into such a stream may prove the means of spreading disease. All streams which are not offensive to the eye or to the smell are liable at times to be used for drinking. They are also used for bathing, and this use has been productive of illness. There is also danger of such water being used in connection with articles of food and drink, as for the washing of milk-cans and vegetables. Water-cress is also grown in polluted streams. The taking of shell-fish from such waters has also been shown to be dangerous. Wells in the neighborhood of such streams may become contaminated. Whether the use of such water is dangerous to cattle is still a question. The flood water too may be deposited upon the bank, leaving foul suspended matter and sewage bacteria. In his final summary he notes: (1) Both bacteria bed processes and land processes can yield effluents seemingly nonputrescible. (2) In general the effluents from bacteria beds and from land are not to be thought of as safe in the case of drinking-water streams. (3) Chemical standards are essential in the case of potable and nonpotable rivers. (4) A bacteriologic standard is most certainly indicated in the case of drinking-water streams, and is of more importance than the chemical one. (5) A bacteriologic standard is of secondary importance in the case of nonpotable streams, but may prove useful as an adjunct to the chemical standard. Further experiments upon rodents, detailed in the same report, demonstrate the presence of B. anthracis, B. pseudo-tuberculosis, and B. pyocyaneus in the water of certain polluted streams, with a strong probability that B. anthracis was present in a septic tank effluent at Leone. As a result of further experiments by Bryce[1] and others, published in the same report, they conclude: That when an effluent containing upon an average 800,000 bacteria per cubic centimeter, and 10,000 B. coli per cc., is passed through a depth of 4 feet of soil at a slow rate (viz., between 10 and 4 liters per 12 hours), there is a very great reduction in the total numbers and in the B. coli, and the chances of a very pathogenic bacterium like the B. typhosus appearing in the filtrate must be exceedingly small. When it is remembered that normally B. typhosus is not present in the excreta of all cases of typhoid, while B. coli is always present in the feces, and that the proportion of typhoid cases to the total number of the population is not large, that typhoid excreta are usually disinfected, and that it has not been shown that favorable conditions exist in sewage for the multiplication of the typhoid bacillus, it appears evident that land or other method of efficient filtration must totally remove this organism from a Dibdin effluent, did it happen to be present. In these experiments B. typhus was mixed with the effluent flowing upon the filter in far larger proportion than would ever occur in nature, and yet it could not be found in the filtrate.

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[1] Chabal: Filtration des eaux par le sable. Rev. d'hygiène, June, 1902, p. 547.

[2] Second Report of Commissioners upon Treating and Disposing of Sewage, London, 1902.

BURIAL OF THE DEAD.

Premature Burial.—Brouardel[2] says: "Can an individual, after being buried while in a state of apparent death, come to life in the grave? When I read of such a case in the newspapers, I make an inquiry, as Bouchut, Tourdes, and Asmaingaud used to do; I write to the mayor or to the medical officer of health (officier de santé) and ask him if the facts are genuine. The reply I am wont to receive is that the story is quite unknown in that part of the country whence it is supposed to have come, or that the person mentioned is not dead and has not been placed in a coffin."

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[1] Ibid., p. 89.

[2] Death and Sudden Death, 1902, p. 40.

French Regulations Relative to Embalming.—These regulations[1] prescribe that a vial containing the same substances that have been used in the process shall be placed by the side of the body.

Cremation in Japan.—Cremation was introduced into Japan[2] about 1871, and has made rapid progress since that time. Most of the cities have established crematories for this purpose. In Tokio in 1897, 15,000 bodies, or 40% of all who died, were cremated. The same city has seven crematories. The mode of combustion is the same in all. The time required for a cremation is from 3 to 5 hours.

MUNICIPAL HYGIENE.

Tenement-house Reform.—The report of the Tenement-house Commission, which was made to the Legislature of New York in 1901, contained the following summary of the most serious evils of the existing system: (1) insufficiency of light and air due to narrow courts or air-shafts, undue height, and to the occupation by the building or by adjacent buildings of too great a proportion of lot area; (2) danger from fire; (3) lack of separate water-closet and washing facilities; (4) overcrowding; (5) foul cellars and courts, and other like evils, which may be classed as bad housekeeping. The first three might be remedied by construction, but the immediate result of seeking to enforce them at the present time, in the case of new tenements, would probably be to prevent their erection by making them commercially too costly; and as respects the old tenements, to vacate many of them for living purposes, because of the cost of alteration. Reform which went so far as to put a stop to building new houses would harm most the very persons it sought to aid. The Commission outlined the following course of action for the solution of the tenement-house problem in New York: First, to provide proper types of tenement-houses for the future by means of adequate restrictive legislation, and to forbid the erection of any others. Second, to remedy the errors of past years by altering and improving old tenement-houses so as to make them fit for human habitation. Third, to maintain present and future tenement-houses in sanitary condition by adequate supervision.

The Moisture of Dwellings.—Coggi[3] purposes to establish a definite maximum limit of the amount of moisture in the walls of dwellings which should be deemed healthful. He advises the method of Glässgen, as modified by Lehmann and Nussbaum, consisting in a determination in the loss of weight sustained by portions of masonry, when subjected to the action of dry air at a temperature of 100° to 110° C. Eighty-four specimens of mortar were examined, 66 of which were taken from buildings erected within 6 months. The mortar from old houses did not furnish more than 3 % of its weight in water. Houses built within 6 months gave quantities varying from 1.03 % to 12.6 % of water in the mortar. Rossi had previously found 1.5 % as a maximum in the wall of old houses.

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[1] Brouardel: Death and Sudden Death, 1902, p. 101.

[2] Jour. d'Hygiène, Paris, Oct. 18, 1900.

[3] Giornale della R. Societa Italiana d. Igiene, 1901, p. 337.

Use of City Refuse for Production of Steam-power.—Mr. W. F. Goodrich[1] makes the following practical comment upon the use of city refuse, garbage, etc., for the production of steam-power: "Less than 20 years ago destructors were adopted solely to get rid of refuse. If it could only be got out of the way, without nuisance, the plant was considered satisfactory. Contrast this with the position to-day, and what do we find? Simply this—that almost invariably every destructor erected now is decided upon with some other definite object in view besides destruction pure and simple. In these days the refuse destructor is boldly associated with electric lighting of towns and districts, sewage-pumping, water-pumping, and other useful power purposes. Why? There is only one answer, and that is because it is now generally recognized that refuse is a valuable asset; that it possesses a calorific value. Now this has come to be recognized, authorities are not content to put the useful heat developed up the chimney; those days have passed. I ask those who still doubt the value of refuse as a fuel to look carefully into the record of contracts for destructors placed during the past year; they will find that the vast majority have been placed with makers of destructors which not only thoroughly destroy refuse, but raise a large quantity of steam therefrom. It will thus be seen that destruction alone does not satisfy. Personally, I am exceedingly suspicious of the nonsteam-raising destructor, and I am rather suspicious of the destructor which only raises a little steam, my reason being that if refuse is burned rapidly and thoroughly at a high temperature of combustion in a well-designed plant, then good steaming results are inevitable."

Heating Power of Ashpit Refuse (Dawson).—The average heating power of the combustible portion of the refuse is as follows:

	CALORIFIC VALUE, OR UNITS OF HEAT DEVELOPED PER POUND OF COMBUSTIBLE.	
	When dry.	When Containing the Average Percentage of Moisture.
Coal	14,000	9,334
Coke	12,000	8,000
Bones and offal	8,000	5,334
Breeze and cinder	6,000	4,000
Rags	5,000	3,334
Paper, straw, fibrous material, and vegetable refuse	3,800	2,534

The Sanitary Aspect of Acetylene Gas.—Masi[2] offers a vigorous defense to the use of acetylene upon sanitary grounds. According to

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[1] The Economic Disposal of Towns' Refuse, by W. F. Goodrich, M. E., London, 1901.

[2] Annali d'igiene sperimentale, 1902, p. 50.

Gréhant, Frank, and Weyl, it does not become dangerous to health until it attains a proportion of 46 % of the air, and requires as much as 76 % to cause death. Its lighting power is from 15 to 20 times that of gas in an ordinary burner. Masi studied the intensity of the flame, the quality of the light, the production of heat, and the alterations of the air which were due to its combustion, in an apartment in the cellar of the Institute of Hygiene at Rome, in the worst possible conditions of ventilation. It consumes less oxygen and evolves less carbonic acid and watery vapor than other illuminants except electricity. It also produces less heat than gas or petroleum, and gives out no ammonia, nitrous acid, sulfureted hydrogen, or carbonic oxid.

VENTILATION.

Composition of the Outdoor Air.[1]—Oxygen, 20.94; nitrogen, 78.09; argon, 0.94; carbonic acid, 0.03; hydrogen and other substances, traces.

Carbonic Acid in Country Air in Scotland.—Volumes of CO_2 per 10,000: April 1 to September 30, maximum 3.58, minimum 2.58, average 2.98; December to January, maximum 3.12, minimum 2.93, average 3.00.

Outdoor Air in Center of London.—Volumes of CO_2 per 10,000: April to September, maximum 4.8; minimum 3.0, average 3.81; October to March, maximum 6.4, minimum 3.0, average 4.22. During a very dense and prolonged fog in London, Russell found as much as 14 volumes of CO_2 per 10,000 in outdoor air.

Ventilation of Factories and Work-shops.—The Parliamentary Committee appointed to consider the question of the ventilation of factories and workshops, in their first report,[2] August 15, 1902, recommended that: (1) Such a standard of ventilation should be prescribed for all classes of factories and workshops not otherwise specially dealt with that the proportion of carbonic acid in the air at about the breathing level, and away from the immediate influence of any special source of contamination, such as a person or light, shall not (except on very foggy days, when no tests should be made, on account of the vitiated state of the outside air) rise during daylight, or after dark only when electric light is used, beyond 12 volumes of carbonic acid per 10,000 of air, and that when gas or oil is used for lighting, the proportion shall not exceed 20 volumes after dark or before the first hour after daylight; the only exception to this rule to be in cases where the extra carbonic acid is produced in other ways than by respiration or combustion, as in breweries, etc. (2) In the case of cotton cloth factories, with artificially humidified air, the occupier should have the option of coming under the regulation just mentioned, instead of remaining under an existing regulation. The remaining recommendations relate to the duties of inspectors; legal proceedings; the analysis of air and the provision of proper apparatus for this purpose. A special form of apparatus for air analysis, devised by Haldane, is described and illustrated in the Appendix.

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[1] First Report of Departmental Committee on Ventilation of Factories and Workshops, London, 1902, p. 93.

[2] London, 1902.

RECENT SANITARY LEGISLATION.

School Hygiene.—California[1] has made it unlawful to require more than 20 recitations a week from students in secondary schools, and to compel any grammar or primary school pupils under 15 years old to study at home. New Mexico provides that persons afflicted with tuberculosis should not be engaged as teachers, and that on complaint, a certificate of health must be provided by a teacher. New Hampshire extended to private and parochial schools the provision that no unvaccinated child who has not had the smallpox shall attend school.

Sanitary Organization.[2]—All the States except Georgia, Idaho, and Oregon now have central sanitary organizations. Montana and Wyoming were added to the list in 1901. New York State has a single Health Commissioner instead of a Board of Health. (Law of 1901, Chap. 29). Laboratories were established in 10 States by the State Boards of Health prior to 1901. An important act was passed in Illinois in 1901 by which local boards of health are "authorized to do all acts, make all regulations which may be necessary or expedient for the promotion of health or the suppression of disease; to appoint physicians as health officers and prescribe their duties; to declare what shall be a nuisance and abate the same; to provide gratuitous vaccination and disinfection; to require reports of dangerously communicable diseases; to require reports of deaths; to issue burial permits when the cause of death is communicable; and to incur the expenses necessary for the performance of the duties and powers enjoined upon the board."

Registration of Vital Statistics.—According to Wilbur, there were in 1901 only 10 States which had anything like a satisfactory system of vital statistics: viz., the New England States, New York, Indiana, Michigan, and New Jersey. A law of 1901 in Illinois (chap. 301) provides for a good system of registration in that State.

Nuisances.—Massachusetts (1901, chap. 427) and Missouri (1901, chap. 73) enacted laws restricting the smoke nuisance and providing penalties for violation. In Virginia a law was enacted (1901, chap. 336) making it a "misdemeanor to expectorate on church floors or aisles or otherwise in a church or other place of worship."

Communicable Diseases.—Laws were enacted in Kansas, Montana, and New Hampshire in 1901 embodying the more modern and generally accepted principles as to the means for combating infectious diseases. In Vermont a law was enacted providing a penalty for using milk tickets a second time, the object being to prevent the transmission of infection by means of such tickets. In Massachusetts (chap. 138, 1901) a law was enacted providing for the prevention of the use of shell-fish from polluted waters. In Montana (1901, chap. 80) the State Board of Health was authorized to adopt measures for general vaccination of the people, and "to prevent the introduction and arrest the progress of smallpox." In

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[1] Dewey's Review of Legislation in 1901, Albany, 1902, p. 80.

[2] Chapin's Digest in Review of Recent Legislation, published by New York State Library, March, 1902.

Utah a backward step was taken since by chap. 18, 1901, vaccination is not to be required of teachers and pupils. In five States isolation hospitals for infectious diseases are required by law, and special laws have been enacted, authorizing cities to build such hospitals. Massachusetts provided for a sanatorium for consumptives by a law of 1895 (chap. 503). New York appropriated $100,000 in 1901 for the same purpose and commissioners have been appointed in New Hampshire, Minnesota, and Rhode Island to consider the subject.

Special Trades.—In several States laws have been enacted providing for the regulation of plumbing and the examination and licensing of plumbers. Michigan and Nebraska have enacted laws of similar character in 1901. In 8 States laws were enacted relative to the examination and licensing of embalmers previous to 1901, and in that year 3 more States were added to the list—Indiana, Michigan, and North Carolina. In 9 States laws have been enacted regulating the barber's trade since 1897.

Food Legislation.—The present tendency of food legislation is to forbid the addition to food of substances injurious to health, and to require the proper labeling of articles which are mixed, compounded, or of inferior quality, so that the labels shall properly describe their ingredients and state whether they are artificially colored or treated with preservatives. In South Dakota a food and dairy commissioner was created by statute (1901, chap. 131), and in Delaware, Illinois, Minnesota, and Missouri amendments were made to the oleomargarine laws. Indiana passed a law forbidding the use of formaldehyd and other injurious preservatives in food. Wisconsin, Minnesota, and Massachusetts also enacted laws in 1901 restricting the use of preservatives. Laws were also enacted in the same year in Minnesota and Massachusetts providing for the labeling of baking-powders.

English Sanitary Legislation in 1900 and 1901.—"With the nation's attention occupied by a great war, the year 1900 was not marked by any specially important legislation affecting the public health."[1] The only sanitary law of any importance enacted by the British Parliament in 1900 was the "Housing of the Working Classes Act " (63 and 64 Victoria, chap. 59), which deals with the provision of working-class lodging-houses, and is intended to simplify the methods adopted for making such provision, by amending a previous act of the same character of 1890. The principal sanitary laws of 1901 consisted in an amendment to the Isolation Hospitals Act (1 Edward VII, chap. 8) making provision mainly for the economic management of such hospitals, and providing that the authorities of one district may care for the sick in another district when the circumstances render it desirable. and the Factory and Workshop Act (1 Edward VII, chap. 22), which consolidates and amends earlier acts. While making definite and specific provisions relative to the sanitary conditions of factories and workshops, it also provides a decided innovation of an excellent character in the direction of disease notification. Hitherto the principle of notification has been confined to infectious diseases, upon the theory that these diseases, being preventable, should be notified to the sanitary authorities; but other diseases besides the infectious class are clearly preventable, hence the law provides that "every medical practitioner attending on or called in to visit a patient whom he believes to be suffering from lead, phosphorus, arsenical or mercurial poisoning, or anthrax, contracted in any factory or workshop, shall send to the Chief Inspector of Factories at the Home Office in London a notice stating the name and full postal address of the patient and the disease from which, in the opinion of the medical practitioner, the patient is suffering, and shall be entitled for such notice to a fee of 2s. 6d." Special provisions are also made in the same act for the inspection of bake-houses and laundries.

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[1] Sanitary Record Diary, 1901, p. 17.

VITAL STATISTICS.

Vital Statistics of England for 1901.—The advance sheet containing the general abstract of the marriages, births, and deaths of England for 1901 presents the following statistics, to which the Registrar General adds the following note: "The numbers of births and deaths in this table are obtained from the quarterly returns, furnished by the local registrars, and will probably be found to differ slightly from the revised figures, to be hereafter presented in the Annual Report." Population of England and Wales, estimated to the middle of 1901, 32,617,001. Marriages, 259,082 (persons married, 518,164); births, 929,270; deaths, 551,316. Marriage-rate, 7.94 per thousand; marriage-rate (persons married), 15.89 per thousand; birth-rate, 28.49 per thousand, death-rate, 16.90 per thousand; sex of births: 473,713 males, 455,557 females, or 1000 females to 1040 males.

QUARTERLY PERCENTAGES. ENGLAND AND WALES.

	MARRIAGES.	BIRTHS.	DEATHS.
First quarter of year	16.90	24.96	26.59
Second quarter of year	27.81	25.13	23.33
Third quarter of year	27.84	25.28	25.26
Fourth quarter of year	27.45	24.63	24.82
	100.00	100.00	100.00

London: Estimated population (1901), 4,543,757. Marriage-rate, 17.61 per 1000 inhabitants; marriage-rate (persons married), 8.80 per 1000 inhabitants; birth-rate, 28.87 per 1000 inhabitants; death-rate, 17.13 per 1000 inhabitants.

Vital Statistics of France.—Drouineau[1] states that the excess of births over deaths in France in 1899 amounted to 31,894, but in 1900 this excess was nearly balanced by an excess of deaths over births, amounting to 25,988, so that the net gain of the population by natural increase in the whole country in the two years was only 5906. The following comparative figures are also given in the same journal. Excess of births over deaths in 1899: France, 31,894; Great Britain, 422,156; Germany, 795,107; Austria-Hungary, 530,806; Italy, 385,165.

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[1] Rev. d'Hygiène, Jan., 1902, p. 69.

Vital Statistics of the German Empire in 1899.—Population in 1899, 54,115,292: Living births, 1,925,924; birth-rate, 35.6 per 1000 inhabitants. Deaths, 1,163,292; death-rate, 21.5 per 1000 inhabitants. Excess of births over deaths, or natural increase, 762,632.

DEATHS AND DEATH-RATES FROM CERTAIN SPECIFIED CAUSES.

Causes.	Deaths.	Death-rates per 10,000 Living.	Minor Causes.	
			Causes.	Deaths.
Tuberculosis	117,419	21.7	Cerebrospinal meningitis	304
Diarrheal diseases of children	165,354	30.5	Smallpox	28
Diphtheria	24,470	4.5	Anthrax	23
Whooping-cough	19,339	3.6	Hydrophobia	11
Scarlet-fever	13,532	2.5	Trichinosis	1
Measles	13,108	2.4		
Typhoid fever	6,001	1.1		
Puerperal fever	3,054	0.56		
Cancer	38,918	7.2		
Suicide	10,418	1.9		

Infant Mortality in France.—Bertillon[1] presents many interesting facts about the mortality of infants supported at the public expense in France. The following table shows the daily number of deaths of this class of infants in 1898, together with those of infants in general:

MORTALITY PER 1000 OF INFANTS UNDER ONE YEAR. FRANCE, 1898.

Ages.	Infants Supported at Public Expense.	Infants in General.
0– 4 days old	0.67	3.75
5– 9 " "	1.38	1.80
10– 14 " "	3.10	1.84
15– 29 " "	2.57	1.23
30– 90 " "	1.16	0.65
91–180 " "	0.71	0.43
181–365 " "	0.39	0.28

By the foregoing table it appears that the mortality of infants supported at the public charge is not so great during the first few days of life as it is in the third five days, while that of infants in general is highest in the first days of life. Bertillon explains this apparent paradox by stating that parents are unwilling to put newborn infants out to nurse or to be weaned when they are apparently at the point of death.

Number of Physicians in Different Countries.—Prinzing[2] estimates the ratio of physicians to the population in different countries as follows. The ratio is expressed per 10,000 inhabitants, and includes surgeons with physicians: Germany, 5.1; Austria, 4.1; Hungary, 2.8;

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[1] Rev. d'Hygiène, Aug., 1902, p. 690.

[2] Zeitschr. f. Sozialwissentch., 4, 7.

Italy, 6.3; Switzerland, 6.1; France, 3.9; Spain, 7.1; Belgium, 5.2; England, 6.1; Scotland, 7.7; Ireland, 5.6; Denmark, 6.4; Norway, 5.3; Sweden, 2.7; Russia (Eur.), 2.7. These figures are for the last ten years. The proportion per 10,000 existing in the large cities is as follows: Berlin (1900), 14.1; Vienna, (1896) 13.0; Buda pesth (1896), 16.4; Paris (1896), 9.7; Brussels (1897), 14.7; London (1895), 12.8; Madrid (1899), 24.4. The number of physicians who practice homeopathy in the home of its birth is surprisingly small. The following figures are for the year 1898. Number of homeopathic physicians and ratio of all practitioners: Wurtemberg, 30, or 3.7 % of all physicians; Prussia, 136, or 0.9 %; Bavaria, 16, or 0.6 %; Saxony, 15, or 0.8 %; Baden, 8, or 0.9 %. Quackery appears to flourish in Germany and has sensibly increased in the past 20 years. Of this class of empirics the figures were as follows in 1898, per 10,000 inhabitants: In Saxony, 16.4; in Wurtemberg, 5.5; in Prussia, 5.3; in Bavaria, 4.5; in Baden and Hesse, 2.9; in Alsace-Lorraine, 1.3. In Saxony, where quackery appears to be most prevalent, the occupations of those in practice were as follows: 44 weavers, 28 stocking-makers, 26 merchants, 24 mechanics, 23 barbers, 23 shoemakers, etc. The author thinks it is high time to take measures to limit the rapid spread of quackery in Germany.

DEATHS FROM DISEASE IN CERTAIN WARS OF PAST CENTURY.[1]

NAME OF WAR.	NATION.	YEAR OR PERIOD.	DEATHS FROM DISEASE PER 1000 STRENGTH.
Walcheren Expedition	Great Britain.	1809	346.9
West Coast of Africa	Great Britain.	1824	690.0
Mexican	United States.	1846–48	100.0
Crimean	Great Britain.	1854	230.0
Chinese	France.	1862	118.0
Civil War	United States.	1862	40.0
Civil War	United States.	1863	60.0
Franco-Prussian	Germany.	1870–71	18.6
Afghanistan	Great Britain.	1878–80	93.7
Soudan	France.	1888-89	280.0
Madagascar	France.	1895	300.0
Chino-Japanese	Japan.	1895	14.8
Spanish-American	United States.	1898	25.0

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[1] Munson, Theory and Practice of Military Hygiene, 1901.

PHYSIOLOGIC CHEMISTRY.

By WALTER JONES, Ph.D., and REID HUNT, M.D.,
OF BALTIMORE, MARYLAND.

A careful chemical and pharmacologic examination of the **active principles in the skin of the toad** (the species was Bufo vulgaris) has been made by Faust[1]; two nitrogen-free bodies, **bufonin and bufotalin,** were isolated. Bufonin was obtained in crystalline form and was found to have the formula $C_{34}H_{54}O_2$. Bufotalin seems to be an oxidation product of bufonin; the provisional formula $C_{34}H_{46}O_{10}$ is proposed for it. Bufotalin has an acid reaction. The physiologic action of these bodies is the same as that of the entire secretion and is very similar to that of digitalis; bufotalin is more active than bufonin, the fatal dose of the former being, for mammals, on subcutaneous injection, 0.0005 gm. per kilo body-weight. Pröscher[2] extracted the skins of Bufo cinereus (the common garden toad) and Bombinator igneus with normal saline solution and obtained a solution of a substance which he calls **phrynolysin,** which has the properties of a toxin; it is hemolytic for the blood of various animals and is destroyed by the digestive ferments, by alkalies, alcohol, ether, and by heating to 56° C. The serums of most vertebrates have no anti-bodies to this substance; rabbits, however, form an antitoxin when treated with the poison for 14 days. This antitoxin also prevents the hemolytic action of the poison upon sheep's blood. Sachs[3] reports experiments with another poison which has the properties of a "toxin"—that of the garden spider (Epiria diadema); he calls the active principle **arachnolysin.** This poison, which was extracted by salt solution, is very active in laking the blood of rats and rabbits; the blood of some other animals is not laked. The stromas of the corpuscles which are dissolved by the poison unite with the poison as required by Ehrlich's side-chain theory. The poison is destroyed by heating to 70° to 72° C. An antitoxin was produced by immunizing guinea-pigs; the serum of such animals protected the blood-corpuscles of the rabbit against the poison. These observations show the toxic character of the poison.

Arsenic.—It will be remembered that Gautier, by means of a special analytic procedure which he himself devised, was able to demonstrate the normal presence of arsenic in the body, but showed it to be localized. The element was found principally in the thyroid gland and to some extent in the thymus gland, the skin, the hair, and the brain, but was always absent from the spleen, the liver, the blood, the urine, and the feces. Gautier even proposed to show that arsenic forms compound proteids in which the arsenic plays a part similar to that of phosphorus in the nucleoproteids, and attempted to trace a connection between the growth of hair and nails, the functions of the genital organs, and the occurrence of arsenic in the thyroid gland. Gautier's experiments are somewhat surprising in view of the countless negative results which are recorded in medicolegal literature, and especially as Ludwig has shown that after ingesting arsenic the poison is principally localized in the liver and held more tenaciously than by any other organ of the body. Hödlmoser[1] has repeated a great deal of his work, using sometimes Gautier's method and sometimes the methods of Fresenius and Babo and others. He shows that by the older methods, 0.7 mg. of arsenic can be recovered from a mixture of 0.9 mg. of arsenic and 100 gm. of liver, and that Gautier's method, aside from its several points of disadvantage, is no more sensitive. In 8 cases he is unable to show the presence of arsenic in the thyroid. Even in the case of the wether and the pig, whose thyroids are claimed by Gautier to contain unusually large quantities of arsenic, he could obtain only the merest semblance of a mirror, no different from that produced with an equal amount of liver, and which Hödlmoser believes is merely a film of carbon due to the decomposition of hydrocarbons formed in Gautier's process. Cerny[2] also finds that there is no regularity in the occurrence of arsenic in the thyroid, it being absent as often as present. In some cases he finds arsenic both in the liver and thyroid of the same individual; in others it is present in the thyroid and not in the liver, while in a third series it is found in the liver but not in the thyroid. Cerny concludes that the variable occurrence of such minute quantities of arsenic in the body is entirely without physiologic significance; that as arsenic is found widely distributed in nature, it is not at all surprising that occasional traces should be found in animal organs.

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[1] Arch. f. exper. Path. u. Pharm., 47, p. 278.

[2] Beitr. zur. chem. Physiol. u. Path., 1, p. 575

[3] Ibid., 2, p. 125.

Bilirubin.—The usually accepted formula for bilirubin ($C_{16}H_{18}N_2O_3$) has served as a basis for many physiologic chemical deductions, especially those concerning the relation of the coloring-matter of the bile to the oxyhemoglobin of the blood. Orndorff and Teeple,[3] however, claim that the formula $C_{34}H_{36}N_4O_7$ more closely represents the composition of the pigment. They purify the bilirubin from 0.75 pound of gall-stones, by crystallization from chloroform and dimethylanilin, but introduce every conceivable precaution both for the elimination of substances insoluble in these solvents, and for the prevention of decomposition of the pigment in process of manipulation. Nevertheless a substance results whose nitrogen analysis falls 0.5 % below that required for the accepted formula. Orndorff and Teeple also report some incomplete studies on the structure of the pigment. Küster[4] believes that bilirubin prepared by the method of Orndorff and Teeple should contain a foreign pigment whose low nitrogen value would account for the low nitrogen obtained. He has not succeeded in isolating the pigment, but states that it is more soluble in chloroform than bilirubin.

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[1] Zeit. f. physiol. Chem., 33, 329.

[2] Zeit. f. physiol. Chem., 34, 408.

[3] Am. Chem. Jour., 26, 86.

[4] Ber. d. d. chem. Ges., 35, 1268.

Carbohydrates.—Nubegert and Wohlgemuth[1] utilize the very convenient insolubility of d-arabinose diphenylhydrazone in alcohol, not only for an improvement on Wohl's[2] synthesis of the sugar from d-glucose, but as a basis of a method for the quantitative determination of arabinose in the urine. In view of the fact that of the countless contributions of Tollens on the estimation of pentoses not one is applicable in the presence of glycuronic acid, the following seems quite an addition to the science of urinology: 100 cc. of urine is treated with 2 drops of 30 % acetic acid and evaporated to 40 cc. on the water-bath. The fluid is treated with an equal volume of hot 95 % alcohol, allowed to stand 2 hours, and filtered. The filtrate is then treated in the warm bath with an excess of diphenylhydrazin. The crystalline hydrazone thus formed is filtered on a Gooch crucible, washed with 30 % alcohol, dried at 80° C., and weighed. The weight of the arabinose is formed by multiplying by 0.4747. Experiments with d-, l-, and r-arabinose in the urine as well as mixtures of glucose, fructose, glycuronic acid, and arabinose gave results which show the method to be practically exact. Newberg and Wohlgemuth[3] use this method in an extended series of experiments on animals (rabbits). They find that no matter how the substances are introduced into the organism, the levo compound disappears to a greater extent than the dextro. When the racemic form was used, a large amount of the dextro appears in the urine, showing again the preference of the organism for a particular configuration; as Fischer had already observed in the case of ferments. Newberg and Wohlgemuth also observe that animals fed on pentose-free food, as also in starvation, will consume relatively more of both stereoisomeric varieties than rabbits under normal conditions, and explain that under normal conditions the organism has at its disposal a sufficient amount of more easily decomposable carbohydrate which protects the arabinose from oxidation. The same line of reasoning may be used to explain why man excretes pentose taken in the food; *i. e.*, they are protected from oxidation by the practically unlimited supply of dextrose present. Experiments on man also show a preference for l-arabinose, since d-arabinose constitutes 60 % of the arabinose excreted after the ingestion of the racemic form. There still remains the difficult question, Why do patients with pentosuria always excrete racemic arabinose free from either optically active variety?

Glycolysis of the Sugars of the Blood and the Formation of Glycuronic Acid.—Lepine and Boulud[4] find that horse's blood possessing the same reducing power as dog's blood is less dextrorotatory, or may even be levorotatory; this is due to conjugated glycuronic acid. If defibrinated dog's blood be kept for an hour at 39° C. while oxygen is passed through, the fermentable sugar disappears and the dextrorotatory power decreases or the levorotatory power increases. If this experiment is performed with the blood of a dog from which the pancreas has been removed, there is no noticeable diminution of the fermentable sugar. If a few drops of chloroform are added to the blood of a healthy dog and allowed to remain for an hour at 39° C., there is a marked diminution of the dextrorotatory power (or an increase in the levorotatory power) and a slight decrease in its reducing power, while some of the fermentable sugar is retained. These results are thought to be due to the formation of conjugated glycuronic acid and the diminution of the glycolysis of the fermentable sugar.

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[1] Zeit. f. physiol. Chem., 35, 31.

[2] Ber. d. d. chem. Ges., 26, 730.

[3] Zeit. f. physiol. Chem., 35, 41.

[4] Compt. rend., 133, p. 720.

Pavy and Siau[1] failed to find any such marked **glycolysis in drawn blood** as described by Lepine and as required by this author's "ferment theory" of diabetes; they found scarcely any glycolysis during the first 2 hours, and even after 4 to 6 hours the disappearance of sugar was not so very marked. Neither could they confirm Lepine's statement that the greater the amount of sugar present, the greater the loss through glycolysis.

Contrary to the statements sometimes made, there is, according to recent investigations of Pavy and Siau,[2] **no formation of sugar in boiled liver.**

Widdicombe[3] sums up his experiments on the **digestion of cane-sugar** as follows: The intestinal mucous membrane inverts cane-sugar, the portions free from Peyer's patches being especially active; acid suspends its activity but does not destroy the enzyme. Lymphatic glands and the saliva have no action on cane-sugar. The gastric mucous membrane and the gastric juice contain an inverting enzyme which acts only in an acid medium. Proteids interfere with the inverting action of dilute hydrochloric acid.

Charrin and Brocard[4] studied the **utilization of hexoses** by the organism by giving a patient mixtures of levulose, galactose, and dextrose, and determining the quantities excreted in the urine; they conclude that levulose is best utilized; then comes galactose and finally dextrose.

Cremer[5] finds that **rhamnose** (isodulcite) does not lead to the formation of glycogen or fat; comparatively small amounts leave the body as such, but much is oxidized, and so spares other food-stuffs. The paper contains many general considerations on carbohydrate metabolism.

It is thought by some that the symptoms of diabetes are due partly to the withdrawal of sugar from metabolism, while others hold that sugar or some derivative of sugar exercises a toxic action upon the tissues of the body; as a contribution to this subject Scott[6] studied the **influence of the subcutaneous injections of dextrose upon metabolism,** and found that when 5 to 7 grams of dextrose per kilo of body-weight was injected into a dog, there was a marked increase in proteid metabolism. The sugar was given subcutaneously in order to get the maximum effect upon the tissues; when given by the mouth, it is rapidly acted upon by the liver, while when injected into the circulation it is rapidly excreted. The author concludes that dextrose, or more probably an acid derivative of dextrose, under such circumstances acts as a protoplasmic poison like phosphorus, etc. After dextrose a smaller amount of the nitrogen is converted into urea; in this respect dextrose acts like diphtheria toxin and sulfonal.

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[1] Jour. of Physiol., 27, p. 451.

[2] Jour. of Physiol., 27, p. 451.

[3] Jour. of Physiol., 28, p. 173.

[4] Compt. rend., 134, p. 48.

[5] Zeit. f. Biol., 42, p. 428.

[6] Jour. of Physiol., 28, p. 107.

Artega[1] makes the interesting observation that in the **phloridzin glycosuria** in cats the ratio of urinary dextrose to the nitrogen was 2.8 to 1; *i. e.*, it was the same as in the rabbit and goat. After the subcutaneous administration of phloridzin to cats the glucosid could be isolated from the urine.

Kossa[2] finds that a **glycosuria** very similar to that caused by phloridzin results when salts of **chromic acid** are administered to animals; the best results were obtained when potassium chromate was injected subcutaneously into dogs. There is no hyperglycemia; the condition is one of "kidney glycosuria."

Some interesting experiments were made by Nebelthau[3] upon the **influence of fever upon the excretion of sugar** in dogs made diabetic by extirpation of the pancreas; when fever was caused by bacterial toxins there was no change in the amount of sugar excreted in the urine. When the animals were infected with the tubercle bacilli, however, there was a diminution in the amount of sugar in the urine.

A summary of recent work on the clinical aspects of acetone, aceto-acetic acid, and β-oxybutyric acid is given by Müller.[4]

In a paper published two years ago Alfthan[5] stated that he had found Landwehr's **"animal gum"** (this is probably a mixture of substances precipitable by alcohol) **in the urine of cases of diabetes mellitus** in amounts varying from 1.2 to 36.9 gm. per day; 0.44 gm. was found in normal urine. In the present paper the author reports on the examination of the urine in two cases of diabetes insipidus; in both cases the animal gum was diminished rather than increased.

Alfthan[6] gives the following method for the **detection of pentoses in the urine**: 500 cc. of the urine is benzoated; the esters so formed are decomposed by sodium ethyl and the solution filtered; the filtrate is tested for pentoses by phloroglucin and hydrochloric acid, or by orcin and hydrochloric acid. Glycuronic acid does not pass into the filtrate.

Colloids.—Pauli and Rona[7] have a long paper on the changes in the **physical condition of colloids**; the effects of various substances (both electrolytes and nonelectrolytes), the electric current, etc., upon the temperature at which gelatin "sets," melts, etc., are discussed at length.

Moore and Parker[8] contribute a comprehensive paper on the **osmotic properties of colloidal solutions.** The points discussed are so numerous that the reader must be referred to the original; among the conclusions reached, however, the following may be mentioned: A definite osmotic pressure is exerted by colloids in solution, and there must be corresponding variations in the freezing- and boiling-points, but these would be so small as to fall within the limit of experimental error. The osmotic pressure cannot be used for determining the molecular weight of colloids, since it gives in cases of substances of known molecular weight, such as soaps, apparent molecular weights which are 20 to 60 times too large; the molecules are present in the form of "solution aggregates," and when the number of molecules present in such aggregates is sufficiently large, the latter begin to be visible by interfering with the light rays or may appear as granules. The solution aggregates form single osmotic units. The weight of the solution aggregate varies with conditions of the solutions; it is 4 to 5 times as great for serum-albumin as for egg-albumen, and in the case of the former it is reduced to about one-fifth its value by alkalization. The authors think that the osmotic pressure of proteids does not take any share in lymph-formation or absorption, and also that the "cells of the membrane in the glomeruli of the kidney probably act as secreting structures; for the differences in pressure are probably not sufficient to cause pressure filtration of a proteid-free filtrate, and are certainly many times too small to separate carbohydrates in this manner."

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[1] Am. Jour. of Physiol., 6, p. 173.

[2] Arch. f. d. ges. Physiol., 88, p. 627.

[3] Arch. f. exper. Path. u. Pharm., 46, p. 384.

[4] Fortschritte der Med., 20, p. 527.

[5] Berl. klin. Woch., 39, p. 162.

[6] Arch. f. experm. Path. u. Pharm., 47, p. 417.

[7] Beitr. z. chem. Physiol. u. Path., 2, p. 1.

[8] Am. Jour. of Physiol., 7, p. 261.

A simple method of making quantitative **determinations of the total sulfates in the urine** is described by Folin,[1] and Garratt[2] describes a method for the **estimation of sodium and potassium in the urine**; for the details of these methods the original articles must be consulted.

Enzymes.—Not the least interesting property of enzymes is their selective action. A given enzyme exerts its activity toward one class of chemical compounds, but is inert toward others, so that when any new product of cell action is found capable of exerting several kinds of enzymic activity we are accustomed to look to an analytic separation which will show the presence of several enzymes. This selective action stands in opposition to the explanation that enzymes are simply finely divided forms of matter or that their activity is to be attributed to surface tension. Cohnheim[3] finds in the mucous membrane of the dog's intestine an enzyme which is without action on native proteids, but which decomposes proteoses with the greatest ease. The presence of this enzyme, which Cohnheim calls **erepsin,** explains the well-known disappearance from the intestine of the difficultly diffusible proteoses and peptones. The syntonin obtained from a kilo of beef-muscle was submitted to an artificial gastric digestion for 30 days. The solution of proteoses and peptones thus obtained was made faintly alkaline, an erepsin solution from the dog's intestine was added and the digestion allowed to proceed for 2 weeks. At the end of this time the complete disappearance of proteoses and peptones was shown by the failure of the product to respond to the biuret reaction. As Hart[4] had already made a very close examination of the hydrolytic products of syntonin under the influence of boiling sulfuric acid, a comparison could easily be made. Hart found the nitrogen of the ammonia to be 7.13 % of the total proteid nitrogen. Cohnheim found 7.20 %. Among the products Cohnheim found histidine, lysine, and arginine, though in somewhat different proportions from those of Hart, and in the filtrate from the phosphotungstic acid precipitate he was able to show leucin and tyrosin. No examination was made for other monamido acids. Cohnheim naturally believes that proteids are completely split into these crystalline products before absorption occurs. As erepsin exerts no action on native proteids (except casein), it cannot be identical with trypsin. The enzyme digests all proteoses and peptones to the point where the biuret reaction fails. Protoproteose and denteroproteose disappear quickly. Heteroproteose and antipeptone soon lose the biuret reaction to a trace, but the removal of this last trace requires a long continued digestion. Erepsin is without action on the proteids of the intestinal wall even though the digestion is prolonged to 6 weeks, nor does it exert hydrolytic influence on the Bence-Jones proteid. This curious proteid seems, therefore, not to be a proteose. On the other hand, the ease with which the casein of milk is attacked gives additional evidence that this proteid is structurally very different from other proteids. It is interesting that the food of infants (*i. e.*, one constituent) can be digested in the absence of both pepsin and trypsin. Erepsin digests protamine with great ease and histone with difficulty, two facts which might have been inferred from the positions of these two substances in a chemical classification.

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[1] Am. Jour. of Physiol., 7, p. 152.

[2] Jour. of Physiol., 27, p 507.

[3] Zeit. f. physiol. Chem., 35, 134.

[4] Zeit. f. physiol. Chem., 33, 347.

Following closely the work of Nencki and Sieber,[1] Pekelharing[2] makes a most interesting contribution on **pepsin.** He finds that gastric juice obtained by Pawlow's method always carries in suspension a phosphoproteid and that by filtering the gastric juice immediately after obtaining, it is possible to prepare phosphorus-free specimens of pepsin. The filtered gastric juice was dialyzed against distilled water for 20 hours at a temperature just above the freezing-point, until the acidity was reduced to about 0.02 % hydrochloric acid. While this causes a precipitation of most of the pepsin, the part which remains in solution is purer and can be salted out by half saturation with ammonium sulfate. Pekelharing found that preparations obtained in this way are extremely active, yet no trace of phosphorus could be shown with 0.6 gram of the substance, a discovery impossible to reconcile with the opinion of Nencki and Sieber that the molecule of pepsin contains a lecithin group. Pekelharing's preparation contains 0.49 % of chlorin; that of Nencki and Sieber 0.47 % to 0.48 %. Pekelharing dissolved his substance in 1 % oxalic acid and dialyzed until the pepsin was again precipitated. The chlorin should be removed unless it is in chemical combination, yet the preparation contained chlorin and digested actively when dissolved in oxalic acid. The analytic results obtained with these phosphorus-free specimens accord closely with the composition of a simple proteid. The mean of several analyses was:

$$C = 51.99 \qquad H = 7.07 \qquad N = 14.44 \qquad S = 1.63$$

The ash-content was very low; from 0.1 % to 0.2 % in which iron was shown. The substance is levorotatory—$(\alpha)_D$ = about 50°—and appears under the microscope as transparent balls, which are not doubly refracting; crystals were never obtained. When an acid solution of these preparations is suddenly heated, a precipitation occurs which Friedenthal, and subsequently Nencki and Sieber, showed would yield a pentose on hydrolysis (these experimenters having obtained their coagulum directly from gastric juice). Pekelharing also showed that pepsin from the pig gives xanthin on hydrolysis. In spite of these points of resemblance, Pekelharing refuses to place pepsin among the nucleoproteids on account of its failure in phosphorus. Pekelharing heated 75 cc. of gastric juice suddenly and cooled. The coagulum was filtered off, washed with alcohol and ether and dried. This product was treated with 1 % caustic potash on the water-bath and the solution acidified with hydrochloric acid, when sulfureted hydrogen escaped and a fine white precipitate occurred. This was filtered off. The filtrate gave pentose when boiled with hydrochloric acid. The precipitate, which Pekelharing calls pepsinic acid, is soluble in hot alcohol. It is insoluble in water, soluble in alkalies, gives the proteid reactions, but yields no sulfureted hydrogen on boiling with alkalies. The same product, pepsinic acid, was prepared from stomach mucosa of the pig and from phosphorus-free pepsin of the dog. Pekelharing's principal reason for believing that his preparation is pepsin and not a proteid contaminated with pepsin is as follows: By injection of alcohol into the rectum during the feeding the secretion is greater in amount, a little richer in hydrochloric acid, but poorer in digestive ferment. Yet the digestive force of the secretion is always proportional to the amount of pepsinic acid which the secretion will yield. Pekelharing subscribes to the view of Nencki and Sieber that several enzymic acts may be inherent in one and the same molecule, for his pure phosphorus-free pepsin digests proteids, coagulates milk, and produces the plastein coagulum in solutions of proteoses. Although the gastric juice used exhibited lypolytic activity, the pepsin preparation had no influence on fats. Pekelharing does not regard the fact that active pepsin solutions have been prepared which show no proteid color-reactions as any evidence against the view that pepsin is a proteid, for this could easily be explained by assuming that the digestive activity of pepsin is far more sensitive than the proteid color-reactions.

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[1] Zeit. f. physiol. Chem., 32, 291.

[2] Ibid., 35, 8.

Bellamy[1] reports a number of experiments upon dogs on **trypsin and its zymogen** in which he brings forward some new facts in support of the Schiff-Herzen hypothesis of the relation of the spleen to the formation of trypsin. Briefly stated, his views are as follows: Normally an internal secretion passes from the spleen to the pancreas at the time when gastric digestion is active; this secretion causes the tryptic zymogen to be converted into trypsin, which then appears in the glandular ducts. After the removal of the spleen the pancreas is in a condition of complete and permanent atrypsia; the pancreatic juice, however, still contains the zymogen which may be converted into trypsin in the intestine through the agency of some product of the small intestine. Pawlow had already shown the presence of a substance, especially plentiful in the jejunum, having this action upon the pancreatic zymogen. Some of the **condi-**

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[1] Jour. of Physiol., 27, p. 323.

tions of conversion of the pancreatic zymogens in extracts into enzymes are discussed at length by Vernon.[1] One of the conclusions reached is that the enzyme itself is one of the most important agents in this conversion; this seems to be the explanation of the fact that a pancreatic extract may remain comparatively inactive for a number of days and then rapidly develop its proteolytic power. As soon as a small quantity of "active" enzyme is formed it brings about the conversion of some of the zymogen; the enzyme so formed converts more zymogen, and so on. An active pancreatic extract of one animal will render an inactive extract of the pancreas of an animal of another species active. The amount of free enzyme in a gland was slightly increased by keeping the gland for 20 hours before extraction or by treating it with 0.2 % acetic acid or with small quantities of tryptic digestion products. The bubbling of oxygen or carbon dioxid through an inactive extract had little effect; if, however, a little active extract was added, oxygen increased while carbon dioxid decreased the amount of enzyme liberated.

Pancreatic Diastase and its Zymogen.—Vernon[2] finds that extracts (aqueous, alcoholic, saline or glycerin) of the pancreas of various animals (including man) increase in diastatic activity if kept in contact with the tissue for some days; extracts filtered off from the gland show no such increase in activity. These results are considered to show that pancreatic diastase exists in the gland in the form of a zymogen which is converted into the active enzyme.

From an extensive study of various **diastases** Vernon[3] concludes that malt diastase is an essentially different body from animal diastase; that salivary diastase is different from pancreatic, and that probably the pancreatic diastases of various animals are different bodies.

Herzog[4] reports an extensive series of experiments on various animals to determine whether the **pancreas contains an enzyme which is capable of splitting dextrose** into alcohol and carbon dioxid; his results were negative, but the author does not seem to be satisfied that the hypothesis of the existence of such an enzyme is disproved.

Benedict[5] studied the **excretion of nitrogen** during nervous excitement; two experiments were made upon an individual who was suffering from the excitement due to the witnessing of foot-ball matches. No distinct changes occurred in the nitrogen metabolism.

Fats.—Kraus and Sommer[6] contribute a short article on the **transference of fat in phosphorus-poisoning;** their chief conclusion is that in phosphorus-poisoning there is a transference of fat to the liver from other parts. Their experiments were made upon mice, and their results are given in two tables. In the first table are given various figures obtained from the examination of normal mice which had been kept on different diets. The animals varied in weight from 24 to 14 gm.; the percentage of fat varied from 29 to 14; the liver made up about one-nineteenth of the total weight of the animal and the percentage of fat in it varied from 12 to 5. About 2 % of the total fat of the body was contained in the liver. In the second table are given the figures obtained from mice poisoned by phosphorus. The weight of these animals had fallen from 20–16 gm. to 14–12 gm.; the fat varied from 8 % to 4 %. The livers had increased in weight until they constituted one-ninth of the total body-weight and they contained from 37 % to 7 % of fat; from 20 % to 44 % of the total fat of the body was contained in the liver. While both the absolute and relative amount of fat in the liver was much increased the total amount of fat in the body had decreased—a fact which is hard to reconcile with the old view of the new formation of fat from the nonoxidation of a nonnitrogenous moiety of the proteid molecule.

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[1] Ibid., l. 269. [2] Jour. of Physiol., 28, p. 136. [3] Jour. of Physiol., 28, p. 156
[4] Beitr. zur chem. Physiol. u. Path., 2, p. 102.
[5] Am. Jour. of Physiol., 6, p. 398. [6] Beitr. zur chem. Physiol. u. Path., 2, p. 86.

Loevenhart[1] has continued his studies on **lipase** (see abstract of last year). "Lipogenesis" is compared with "glycogenesis"; the two phases of lipogenesis are brought about by the same enzyme, which may be fat-splitting or fat-forming according to the conditions. The author describes experiments upon dogs poisoned with phosphorus, and found that the fatty changes were not due to changes in the distribution and amount of lipase in the tissues.

Rosefeld[2] made a study of the **origin of fat** in a number of classes of animals; he showed that in various fishes the fat of the body could be altered by altering the fat of the food. The feeding of carbohydrates to fish led to the formation of a solid fat poor in olein. The relation between the fat of the body and of the food was studied for other classes of animals (cetaceæ, crustaceæ, etc.); the author failed to find evidence for the view that fat is formed from proteids.

Owing to their supposed medicolegal importance, the so-called **"Florence crystals"** have been the subject of numerous contributions. Florence obtained these crystals by treating an aqueous extract of semen with Lugol's solution. They bear a close resemblance to Teichmann's crystals, and were at first thought to be formed characteristically from semen, but several investigators have shown that they may be formed from a variety of objects, although no one has conclusively shown the chemical identity of the substance necessary for their formation. Bocarius[3] describes a method of preparing the crystals in quantity and preserving them. He uses the semen of men and horses, the liver and brain of men, and the liver of the ox. All of these tissues yield the crystals equally well. From these crystals Bocarius was able to prepare a substance which forms a platinum compound of characteristic orange prisms containing 31.6 % of platinum and which forms trimethylamin when heated. Florence crystals are therefore formed from cholin.

Strauss and Wolff[4] studied the **hemolytic action of various serous transudates and exudates** (cerebrospinal and ascitic fluids, pleural and pericardial exudates, etc.) and found that the inflammatory exudates possess greater hemolytic action than do the transudates; the latter are also for the most part, inferior in power to blood-serum. There seems to be some relation between the amount of proteid present in a liquid and the hemolytic power; thus the transudates, which contain very little proteid, have the smallest hemolytic action. Ascitic fluid, however, is an exception; it is rich in proteid but has little hemolytic action. Hence there are factors other than the amount of proteid involved.

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[1] Am. Jour. of Physiol., 6, p. 331.

[2] Münch. med. Woch., 49, p. 17.

[3] Zeit. f. physiol. Chem., 34, 339.

[4] Fortschritte der Med., 20, p. 1.

Carré and Vallée[1] find that there is a close parallelism between the **toxicity of normal blood-serums** and the hemolytic action of such serums. Thus the serum of the horse's blood is not hemolytic for the blood of the guinea-pig; neither is it toxic for the guinea-pig. The reverse is true in the case of ox-blood. This parallelism goes so far that it is possible to determine the toxicity of a serum from its hemolytic activity. When the serum of ox-blood is allowed to act upon the blood-corpuscles of the guinea-pig, the former loses its toxicity and can be injected into the peritoneal cavity of a guinea-pig without causing death; on the other hand, if the ox-blood serum be injected into the peritoneal cavity of a guinea-pig the animal will die, but the serum loses very largely its hemolytic and toxic properties.

From the experiments of Gnezda[2] it seems probable that the **indoxyl** found in the urine is not wholly derived from the putrefactive processes occurring in the intestine for an examination of the urine of patients suffering from various diseases showed that there was a definite ratio between the amounts of indoxyl and urea excreted; the ratio was approximately 1 part of indoxyl to 6 parts of urea. This fact makes it probable that part of the indoxyl is produced by the oxidation of proteid.

Iron.—Delamare[3] finds the **iron of lymphatic glands** of various animals to vary in amount from mere traces to 0.58 per 1000. In inanition it diminishes, while after splenectomy it increases. These facts point to a hematopoietic function in lymph-glands.

Bielfeld[4] records some observations upon the **iron content of the normal human liver**; the most interesting result was the demonstration that the liver of the female contains, as a rule, less iron than does that of the male, and that the content of the former is much more constant than that of the latter. Thus the liver of the female contained from 0.05 % to 0.092 %; that of the male from 0.048 % to 0.367 %. The livers of individuals between the ages of 20 and 25 years contained the smallest amount of iron, and at this period there was no difference between the sexes in the amount present.

Malignant Tumors.—When carcinomatous tissue is kept for some days at the ordinary temperature and putrefaction prevented by the addition of chloroform, the coagulable proteids decrease in amount while the noncoagulable nitrogenous substances increase; in other words, autolysis occurs. Petry[5] finds leucin, tyrosin, lysin, and hypoxanthin among the products of this autolysis. This autodigestion is much more marked than is that in the normal mammary gland. [The carcinomatous tissue was obtained mainly from the breast.] Products of proteolysis were not found in the fresh tumor or in the blood of a patient suffering from a rapidly growing carcinoma. Neither extracts of the fresh carcinoma nor of a carcinoma which had undergone autolysis had any distinct effect upon nitrogen metabolism when injected into a dog.

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[1] Compt. rend. Soc. Biol., 54, p. 176.

[2] Compt. rend, 134, p. 485.

[3] Compt. rend. Soc. Biol., 53, p. 897.

[4] Beitr. zur chem. Physiol. u. Path., 2, p. 251.

[5] Beitr. zur chem. Physiol. u. Path., 2, p. 94.

Emerson[1] finds that when a small amount of fresh carcinomatous tissue is added to an artificial gastric digestion there is a marked diminution of free HCl; the diminution of free HCl was much less marked if the carcinomatous tissue was first heated. The products of this digestion were for the most part carried beyond the albumose stage and were of a basic nature. From these experiments and from analyses of the stomach-contents of patients suffering from carcinoma of the stomach the author concludes that carcinomatous tissue contains an autolytic ferment which digests proteid with the formation of basic bodies combining with HCl. Such combinations have, however, an acid reaction, so that the acidity of the stomach-contents may be high and yet contain no free HCl.

Manganese.—According to the investigations of Harnack,[2] the **absorption and excretion of manganese** is very similar to that of iron; *i. e.*, it is absorbed by the intestinal tract and then excreted, to a large extent, through the same channel. It is not excreted at all, or only in minimal amounts, in the urine. The manganese, in both organic and inorganic forms, is absorbed by the intact intestinal canal. When a permanganate is injected subcutaneously, it is quickly changed into insoluble compounds, which are then partly absorbed and excreted in the feces.

Rettger[3] observed that when **milk** is heated above 85° C. **a volatile sulfid** was liberated; this is probably hydrogen sulfid. Alkalies and alkaline phosphates favor the liberation of the sulfid, while acids and acid phosphates retard it. Hence the amount of sulfid liberated depends largely upon the reaction of the milk. The author thinks his results indicate proteid decomposition and that they may perhaps explain the injurious effects of heating milk.

Nutritive Values.—Prausnitz[4] records experiments on the **nutritive value of meat** and **meat preparations** in man; the most important conclusion is that fresh meat cooked in the usual way is much more easily digested than are most of the preparations in which drying of the meat is part of the method employed. Roast fresh meat is almost entirely absorbed and leads to only a small formation of feces. After most of the various patent preparations the amount of feces was large and contained much unused nutriment. The author emphasizes the fact that the feces consist not only of undigested food, but also represent the excretion of the intestinal walls.

The **nutritive value of gelatin** is discussed by Krummacher.[5] It was found that 72.35 % of the total energy of gelatin could be utilized by the body. When dogs were fed on gelatin, the proteid decomposition was 62.6 % of that which is broken down during inanition; this probably represents the maximum action of gelatin. In a man of average body-weight the amount of proteid which is broken down per day is about 70 gm. If gelatin is given to exert the maximum effect, it being assumed that the same relationship holds for man as for dogs, 33 gm. of gelatin will reduce the proteid decomposed to 56 gm., or 33 gm. of gelatin will spare 14 gm. of proteid.

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[1] Deut. Arch. f. klin. Med., 72, p. 415.

[2] Arch. f. exper. Path. u. Pharm., 46, p. 372.

[3] Am. Jour. of Physiol., 6, p. 450.

[4] Zeit. f. Biol., 42, p. 377.

[5] Zeit. f. Biol., 42, p. 242.

Embden and Glaessner[1] studied the **formation of phenyl-sulfuric acid** in the body by transfusing various organs with blood containing phenol. The liver formed phenyl-sulfuric acid in considerable quantities, but some of the phenol was bound in other ways—probably with glycuronic acid. The intestines and muscles formed no phenyl-sulfuric acid and the lungs and kidneys but traces.

Some authors state that **phosphoric acid is excreted by the kidneys,** while others maintain that it is largely eliminated by the intestines; Bergmann[2] finds that the results depend very largely upon the kind of animal used in the experiment. In dogs there was no excretion of phosphoric acid in the intestine; this was true also when large quantities of calcium salts were administered with the phosphoric acid. In herbivora (sheep), on the other hand, nearly all the phosphoric acid was excreted into the intestine. This difference was also true when the phosphoric acid was given in organic combination (glycerin phosphoric acid); in the dog the phosphoric acid appeared in the urine; in the sheep, in the feces.

Folin and Shaffer[3] studied the **phosphate metabolism** of a patient suffering from a form of depressive insanity; the patient's condition alternated with absolute regularity from day to day, being one day almost normal, while quite disturbed on the other. On the "bad days" the phosphates of the urine were increased. This alteration of greater and less excretion of phosphate was not dependent upon differences in the absorption due to changes in the acidity of the gastric juice, for the administration of hydrochloric acid and sodium bicarbonate was without effect. The authors offer the following hypothesis to explain their results: There exists in this patient on every second day a condition somewhat analogous to that of diabetes, in virtue of which the system or some part of it (presumably the nervous system) is unable to assimilate a part of the phosphate absorbed from the digestive tract; this nonassimilated phosphate is eliminated the same day. On the other days less phosphate is eliminated because the tissues are then repairing the loss sustained on the previous day.

Proteids.—Jolles' work on the formation of urea by the oxidation of proteids was reviewed in the 1902 volume of the YEAR-BOOK. Schulz[4] now makes a very severe criticism of this work. He objects to the addition of 300 to 400 cc. of permanganate, a cubic centimeter and finally a drop at a time, and confesses that in his repetition of Jolles' work he departed from this time-consuming procedure. He also has difficulty in removing inorganic salts from the oxidation products by repeated evaporation and solution in alcohol, and claims that his final product gave a large amount of ash. After dwelling at great length on several somewhat trivial matters of manipulation, he takes up some of Jolles' analytic errors and shows that correct calculation brings only half as much nitrogen as is required for urea oxalate. It is also shown that the numbers in the tabulation in some instances differ from those given in the analytic work. It has also seemed to us somewhat ludicrous to fill up the page with analytic data when one is reporting the simple analysis of a chemical individual. The finding of an arithmetic error in such work, moreover, does not in general discredit the conclusions drawn from the analytic results. There are hundreds of instances to be found in chemical literature, but the one explanation which is always offered shows how useless is the criticism. Jolles[1] describes his arithmetic confusion as deplorable, but claims that it is simply a case of error in copying. In regard to other points in Schulz's criticism, he simply states that if one expects to obtain his results, one must adhere closely to his directions. We are, of course, prepared to learn that Jolles did not succeed in obtaining urea by the oxidation of proteids, but we are scarcely ready to abandon Jolles' work on the ground that he, or even a moderately trained chemist, is unable to separate urea from inorganic salts and analyze its oxalate.

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[1] Beitr. zur chem. Physiol. u. Path., 1, p. 310.

[2] Arch. f. exper. Path. u. Pharm., 47, p 77.

[3] Am. Jour. of Physiol., 7, p. 135.

[4] Zeit. f. physiol. Chem., 33, 363.

In a paper, largely theoretic, on **proteid metabolism** Gruber[2] insists on the correctness of Pettenkofer's and Voit's distinction between the "organ proteid" which undergoes but little and that a constant change, and the proteid derived directly from the food, which is subject to great variations. Experiments are quoted to show the influence of water in increasing the waste nitrogenous products, and that different proteids after absorption vary in their yield of katabolic products.

The **proteids of the muscle plasma** of various animals have been studied by Przibram[3]; the methods of investigation and the nomenclature used were those of v. Fürth. One of the most interesting facts brought out is that myogen ("myosinogen") is found only in vertebrates; the myogen of the lowest vertebrates (cyclostomes), however, differs from that of the higher vertebrates in that it is not precipitated by sodium salicylate. Myosin ("paramyosinogen") was found in both vertebrates and invertebrates. Soluble myogen fibrin, which in the higher vertebrates is found only some time after death, was found very shortly after death in fishes and amphibians; it is probably present in the living animal. Myoproteid was always found in fishes; it occurs in traces in amphibia and is entirely absent in the Amnioata (reptiles, birds, and mammals).

Tendon mucoid, according to the investigations of Cutter and Gies,[4] has very nearly the same composition as chondromucoid and osseomucoid; the three bodies seem to be intimately related. The tendon mucoids (for tendon contains more than one glucoproteid) contain, on

[1] Zeit. f. physiol. Chem., 34, 28.
[2] Zeit. f. Biol., 42, p. 407.
[3] Beitr. zur chem. Physiol. u. Path., 2, p. 143.
[4] Am. Jour. of Physiol., 6, p. 155.

the average, about 2.3 % of sulfur. The **chemical composition** of **tendinous tissue** is described in detail by Buerger and Gies[1]; they find that 85 % of the dried tendo Achillis consists of collagen (gelatin). Elastin was present to the extent of 4.389 %, mucoid to 3.455 %, and albumin and globulin to but 0.593 %. Hawk and Gies[2] publish an extensive paper on **osseoalbumoid,** a proteid substance obtained in comparatively small quantity from bone after hydration of the collagen; this substance is neither keratin nor typical elastin, although it resembles the latter body. The properties of this substance are very similar to those of chondroalbumoid; both are free of phosphorus in organic combination. Richards and Gies[3] have a long paper on the **chemistry of elastic tissue;** the tissue studied was the ligamentum nuchæ of the ox. Several samples of ligament elastin were prepared and analyzed; all of them contained sulfur, none of which could be split off as sulfid on boiling with sodium hydroxid. On decomposing elastin with acid only very small amounts of nitrogen could be split off as ammonia and hexone bases; among the latter arginin, lysin, and histidin were obtained. The elastin was readily digested by pepsin-hydrochloric acid; elastoses and true pepton (in small quantity) were obtained. The ligament contains mucoid and proteids and a small amount of nucleoproteid. The collagen of the ligamentum nuchæ is identical with that of bone and tendon. Creatin, hypoxanthin, and guanin were also obtained.

According to Tebb,[4] **reticulin,** which Siegfried described as characteristic of reticular tissue, is simply collagen which has been "coagulated" by the reagents he employed (especially alcohol and ether) *plus* proteid and nuclein residues of cells; after treatment with these reagents conversion into gelatin is rendered much more difficult by subsequent boiling with water. The main material of both tendon and reticular tissue is collagen, and the fibers of which these structures are composed are histologically identical.

Proteolysis.—The discovery of an analytic method always marks an epoch in chemical development. Fischer's introduction of phenylhydrazin as a reagent caused one of the most obscure chapters in the field to become one of the clearest, while Kütscher's method of separating the hexone bases was followed by a literature that was almost unreadable. Fischer[5] now introduces a method of separating the hydrolytic products of proteids which not only serves to establish quantitative relations between the products already known, but which has led to the discovery of substances hitherto unknown in this connection. This method consists in treating the material in question with alcohol and hydrochloric acid and submitting the ethers thus formed to fractional distillation under diminished pressure. In certain fractions the isolation and identification of various constituents is easily accomplished by simple methods which are in everyday use, while in others advantage is taken of beautifully crystalline compounds of sharp melting-points which are formed by treating amido-acids with phenylisocyanate.[1] Thus, in the case of alanine:

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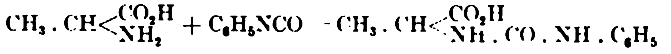
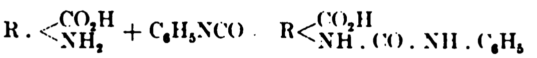
[1] Ibid., p. 216.
[2] Ibid., 7, p. 340.
[3] Ibid., 7, p. 93.
[4] Jour. of Physiol., 27, p. 463.
[5] Ber. d. d. chem. Ges., 34, 433.

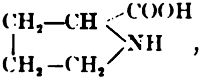


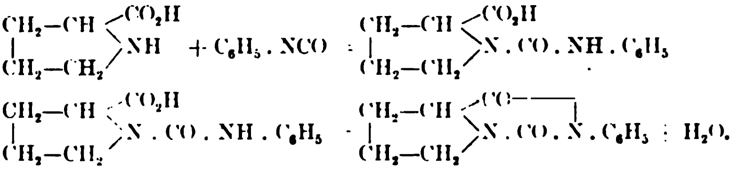
$$CH_3 . CH<\begin{smallmatrix}CO_2H\\ NH_2\end{smallmatrix} + C_6H_5NCO = CH_3 . CH<\begin{smallmatrix}CO_2H\\ NH . CO . NH . C_6H_5\end{smallmatrix}$$

and in general:

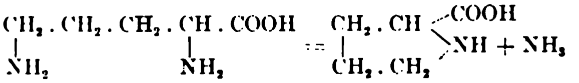


$$R . <\begin{smallmatrix}CO_2H\\ NH_2\end{smallmatrix} + C_6H_5NCO = R<\begin{smallmatrix}CO_2H\\ NH . CO . NH . C_6H_5\end{smallmatrix}$$

Among the hydrolytic products of casein with hydrochloric acid, tyrosin, leucin, aspartic acid, glutanic acid, arginin, and lysin have been proved with certainty. In addition, Cohn[2] has isolated a substance, $C_{12}H_{22}N_2O_2$, isomeric with leucinimid, and probably a secondary product. By the use of the method referred to, Fischer[3] adds: (1) amidovaleric acid; (2) phenyl alanin; (3) α-pyrrolidine carbonic acid. The greatest interest naturally attaches to this optically active pyrrolidine compound,  , which has been hitherto known only as a synthetic product,[4] and in its racemic form, and which furnishes the first instance of a ring compound containing nitrogen as a hydrolytic product of a proteid. This acid, which constitutes about 3.2 % of casein, seems to have escaped the older investigators in this field on account of its great solubility, but Fischer was able to form a difficultly soluble compound, by treatment with phenyl isocyanate, which can easily be converted into an anhydrid melting sharply at 142°.



If the discovery of this acid is to be accepted as the proof of a pyrrol ring in the casein molecule, it is necessary to show that it does not result from the chemical process involved, but is a primary decomposition product. Thus under the action of hydrochloric acid pyrrolidin carbonic acid might be formed from arginin or ornithin by ring closure.



α-δ- diamidovaleric (ornithin) α- pyrrolidin carbonic acid.

Fischer[5] finds, however, that neither arginin nor ornithin will yield α-pyrrolidin carbonic acid under the conditions stated, and that this ring compound can be obtained by hydrolysis of casein with alkalies, a procedure which would prevent ring closure The application of Fischer's method to other proteids has led to results just as interesting as those obtained with casein. Fischer, Levene, and Alders[1] find the following hydrolytic products of gelatin: (1) Glycocoll, 16.5 %; (2) l-pyrrolidin carbonic acid, 5.2 %; (3) l-leucin, 2.1 %; (4) d-glutamic acid, 0.88 %; (5) d-alanin, 0.8 %; (6) aspartic acid, 0.56 %; (7) d-phenylanalin, 0.4%. It will be noted that Fischer's method avoids racemating processes and enables him to isolate the amido-acids in optically active form. In the case of gelatin the ring compound exceeds in quantity the sum of all the amido-acids except glycocoll.

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[1] Paal, ibid., 27, 974.

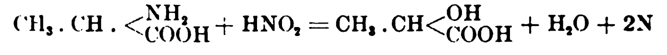
[2] Zeit. f. physiol. Chem., XXIX, 283.

[3] Ibid., XXXIII, 151.

[4] Willstätter, Ber. d. d. chem. Ges., 33, 1160; and E. Fischer, ibid., 34, 454.

[5] Zeit. f. physiol. Chem., 33, 227.

The conclusion of Kühne that a pancreatic digestion of gelatin produces neither leucin nor glycocoll seems to have passed unchallenged since 1877, although it is very generally believed that the application of modern analytic methods would probably place the matter in a different light. Reich-Herzberge[2] finds that after long-continued digestion of large quantities, traces of leucin could be proved. Reich-Herzeberge might very properly have ascribed these traces to proteid impurities in the gelatin, which is usually conceded to be the cause of the faint Millon's reaction given by commercial preparations By treatment with soaps, alkalies, or even hot water, silk may be separated into two constituents: (1) Fibroin, which is the insoluble proteid and forms by far the greatest part of silk by weight; and (2) sericin, a soluble constituent which forms a characteristic property, is often known as silk gelatin. Fischer has made a very close examination of the hydrolytic products of both of these substances. Up to the time of Fischer's publication the hydrolytic products of fibroin conclusively shown were (1) tyrosin, (2) glycocoll, (3) one of the three possible amidopropionic acids. Fischer[3] adds l-leucin and l-phenyl alanin. He also shows that the amidopropionic acid in question is optically active, and by treatment with nitrous acid yields sarcolactic acid.



$$CH_3 . CH . <^{NH_2}_{COOH} + HNO_2 = CH_3 . CH <^{OH}_{COOH} + H_2O + 2N$$

The substance must therefore be identical with d-alanin. While the fibroin of silk was found to yield nonamido-acids in great preponderance, and only traces of hexone bases, the converse was found true in the case of silk gelatin. Both proteids yield serin, the only hydroxy aliphatic acid known to physiologic chemistry.

Several years ago Siegfried was able to prepare from the products of pancreatic proteolysis a substance of the composition $C_{10}H_{15}N_3O_5$, which was practically free from sulfur, and which answered all the requirements of what was then known as antipeptone. This sulfur-free proteid was additionally interesting since Siegfried claimed that it was identical with his then newly discovered carnic acid. But Kütscher's discovery of the hexone bases as constituents of antipeptone threw discredit upon what promised to be a most interesting and important chapter in animal chemistry, and all interest in antipeptone and carnic acid ceased. Siegfried[4] now opens the subject anew with a communication on antipeptone which is extremely interesting, but which does not sustain his former contention. After a very prolonged digestion of the proteid with trypsin all traces of proteose are removed by saturating the solution with ammonium sulfate under various conditions of acidity. By fractional precipitation of the peptone solution thus obtained with iron ammonium alum, and ammonia he is able to isolate two proteids, which he calls α-antipeptone and β-antipeptone. Both substances are snow-white powders, soluble in saturated aqueous ammonium sulfate with the greatest ease. They are ash-free, sulfur-free, have no bitter taste, somewhat soluble in hot alcohol. On cooling, the alcoholic solution deposits transparent microscopic grains. By heating their aqueous solutions on the water-bath proteoses are regenerated and can be salted out with ammonium sulfate. Both proteids give the biuret reaction beautifully, Millon's reaction not at all. Solutions of the hexone bases and of antipeptone were made of the same concentration and equally diluted until the former gave no precipitate with phosphotungstic acid. At this dilution the antipeptone solutions gave heavy precipitates with this reagent. Both varieties of antipeptone are levorotatory, and the β-compound is inverted by boiling with acids. From analyses of the antipeptones themselves, analysis of the barium and zinc salts, and from the results of molecular weight determinations, Siegfried assigns the following formulas: α-antipeptone, $C_{10}H_{17}N_3O_5$; β-antipeptone, $C_{11}H_{19}N_3O_5$. Preparations of both antipeptones (from Witte's peptone) were hydrolyzed with sulfuric acid in the presence of stannous chlorid. Both yield lysin; the β-acid gives also glutamic acid, while the α-compound gives aspartic acid and a base whose properties accord with those of lysatinin. As is well known, Hedin[1] is supposed to have shown that lysatinin is nothing but a mixture of lysin and arginin, but Siegfried[2] is not willing to concede this proof, claiming that lysatinin is a definite chemical individual from which no arginin can be separated. The importance of the matter from Siegfried's point of view is easily appreciated when it is noted that the molecule of arginin contains 4 atoms of nitrogen, while Siegfried's formula for antipeptone contains but 3. The latter could therefore scarcely give rise to the former by simple hydrolysis. Siegfried's position is somewhat strengthened by the results of his work on the antipeptone from gelatin. A molecular weight determination led to the formula $(C_{10}H_{17}N_3O_5)_2$, and on hydrolysis the substance gave a mixture of lysin and arginin. We are prevented from speaking of this work most enthusiastically only by remembrance of a former paper of Siegfried in which the formula $C_{10}H_{15}N_3O_5$ was supported by analysis of various salts, and the substance in question, then called also antipeptone, was shown to be identical in every respect with carnic acid, which itself had the formula $C_{10}H_{15}N_3O_5$.

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[1] Zeit. f. physiol. Chem., 35, 70.

[2] Ibid., 34, 119.

[3] Fischer and Skita, Zeit. f. physiol. Chem., 34, 176; 35, 221.

[4] Zeit. f. physiol. Chem., 35, 160.

In a most interesting contribution Lawrow[3] claims that by a sufficiently **protracted gastric digestion proteids are as completely hydrolyzed as by trypsin,** although the products formed are not the same. Carefully cleansed pigs' stomachs were treated with 0.5 % hydrochloric acid and submitted to autodigestion at the body-temperature for 2 months. Putrefaction was prevented by the introduction of thymol and closing the vessels. On submitting the products to a careful fractional crystallization at least 40 % of the material was found to be crystalline. Lawrow believes that the main part of this crystalline material is a mixture of amidovaleric acid and leucin in molecular quantities, since the product does not change its composition by repeated crystallization and gives analytic numbers that are between the values required for these two acids. He was able to prepare a small amount of leucin in pure condition and to prove the presence of aspartic acid. Tyrosin and the hexone bases were not found. The most curious part of this work is that in spite of the careful provision against putrefaction, pentamethylene diamin and hexamethylene diamin were found in comparatively large quantities, being formed, as Lawrow supposes, at the expense of the hexone bases.

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[1] Zeit. f. physiol. Chem., 21, 298.

[2] Ibid., 35, 192.

[3] Zeit. f. physiol. Chem., 23, 312.

Langstein[1] carried on an **artificial gastric digestion** of 700 gm. of coagulated proteid of horse's serum for 12 months; the following products, in addition to those which have already been obtained in such experiments, were isolated: tyrosin, glutaminic acid, oxyphenyl-ethylamin, a substance belonging to the pyridin series, a polysaccharid containing nitrogen, and an acid belonging to the carbohydrate group and containing nitrogen. Thus long-continued peptic digestion leads to the formation of simple substances similar to those produced in a short time by tryptic digestion. The author gives in detail the methods of separating the above bodies. In a second communication[2] Langstein describes a similar experiment made with crystallized egg-albumen; the same products were obtained, and, in addition, small quantities of cystin and lysin. These results emphasize the close resemblance between the action of pepsin and trypsin; one of the chief points of difference is the fact that with peptic digestion the biuret reaction persists.

Glaessner[3] describes experiments showing a **regeneration of proteid** from its hydrolytic products; this change relates exclusively to the albumoses, and is brought about by the gastric mucous membrane. In the dog this change commences soon after digestion begins, reaches its maximum 5 or 6 hours later, and then subsides. The author thinks that rennin does not play any part in the process, and that the formation of "plastein" by rennin *in vitro* is probably not analogous to what occurs *in vivo*.

Hopkins and Cole[4] have succeeded in isolating that product of pancreatic digestion which gives a red-rose color with bromin-water and which has been called **tryptophane**; its formula is $C_{11}H_{12}N_2O_2$, and it yields indol and skatol on heating. This substance gives, in a typical manner, the Adamkiewicz reaction for proteids. [The authors have shown elsewhere that this reaction depends upon the presence of glyoxylic acid in the acetic acid.]

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[1] Beitr. zur chem. Physiol. u. Path., 1, p. 507.

[2] Ibid., 2, p. 229.

[3] Beitr. zur chem. Physiol. u. Path., 1, p. 328.

[4] Jour. of Physiol., 27, p. 418.

Cassart and Saux[1] record some observations on the **toxic substances produced by peptic digestion.** When an artificial gastric digestion of meat is injected into the circulation of an animal, the animal dies in convulsions; this result is not due to the acid contained in the mixture, but seems to be due to a specific poisonous substance. The poisonous material was not identified chemically, but is thought to be the same as Brieger's "peptotoxin." The authors think that such substances may be formed in natural digestion and may explain such processes as tetany.

A number of physical and chemical data with regard to **Witte's peptone** have been determined by Sollmann[2]; some of these are as follows: The alkalinity (with methyl-orange as an indicator) is just that of dried egg-albumen; the ash varies from 2.4 % to 6 % with various degrees and time of heating; both the freezing-point and conductivity indicate a dissociation, the extent of which varies with the sample. It appears to be monovalent toward acids and bases. The author finds that the peptone has an average molecular weight of 630, but that it is dissociable into 4 molecules by dilution; on heating, an alkaline molecule is split off. In a second paper Sollmann[3] discusses the combination of Witte's peptone with formaldehyd; the characters, solubilities, etc., of the precipitate are described. The work of Koch[4] on the physiologic action of formaldehyd may be mentioned in this connection; working with yeast, Koch concludes that formaldehyd causes the death of the cell by rendering its proteid food-supply useless and not by forming active oxygen (as was suggested by Van 't Hoff) or by destroying the zymase.

Suprarenal.—Two years ago Takamine placed upon the market a microcrystalline suprarenal preparation which he called adarenalin. The preparation was not hygroscopic, failed to give precipitates with the alkaloidal reagents, possessed physiologic activity to a high degree, gave the green color with ferric chlorid in great dilution, and contained phosphorus. As the preparation was practically insoluble in cold water, physicians were advised to use a solution 1 : 1000, which was also placed upon the market. Little could be learned about adarenalin further than the gratuitous information contained in circulars that Takamine had isolated the blood-pressure-raising principle of the suprarenal gland in stable and pure crystalline form. Takamine[5] carefully guarded his method of preparation and gave no clue to the relation of adarenalin to either Abel's epinephrin or von Fürth's suprarenin. He does, however, cite a most remarkable case of polymorphism. Adarenalin is described as crystallizing in 5 different forms, of which probably the most interesting to the crystallographer are the tomato-shape and the boat-shape, since he has probably already heard of rhombic plates, needles, and prisms. Later, Aldrich[2] claims to have prepared a substance identical with Takamine's adarenalin. His work is certainly independent, for Takamine had not up to this time made known his method of preparation, and it will perhaps be impossible to determine which of the two was the first to have the substance in hand. Aldrich[1] heated an aqueous infusion of the gland to coagulate the proteids, separated various impurities with lead acetate, and, after removing the excess of lead with sulfureted hydrogen, precipitated the remaining proteids and salts with alcohol. The alcoholic solution was evaporated under diminished pressure and the adarenalin finally precipitated by the addition of ammonia. From a comparison of the analysis of his preparation with that of Takamine's substance, together with a qualitative study of the two, Aldrich concludes that the two are identical, and assigns the formula $C_9H_{13}NO_3$.

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[1] Compt. rend. Soc. Biol., 53, p. 1072.
[2] Am. Jour. of Physiol. 7, p. 203.
[3] Ibid., 7, p. 220.
[4] Ibid., 6, p. 325.
[5] Therap. Gaz., 1901, 221.

Von Furth[2] describes the preparation of "**adrenalin**" by the use of his characteristic iron compound. His most prized product is a syrup which assumes a solid form only on drying in a desiccator. All attempts to obtain a crystalline product were fruitless, yet he pronounces his preparation identical with the adarenalin of Takamine. Surely one has only to dissolve a substance that is identical with adarenalin in hydrochloric acid and add ammonia, when a crystalline substance will be obtained. We stated in substance, in the last edition of the YEAR-BOOK, that von Fürth seemed to lack enthusiasm in regard to his original point of contention. He now makes the acknowledgment (and we consider it the most valuable part of his paper) that he cannot adhere to his "provisionally (?) expressed" opinion that adarenalin is either dihydro- or tetrahydro-dioxypyridin. Abel[3] finds that it is possible to split off benzoic acid from members of his epinephrin series, and attributes this property to the retention of a benzoyl group which it had been found impossible to remove by any but destructive methods. As the compound which he has called epinephrin is really a monobenzoyl derivative of the formula $C_{17}H_{15}NO_4$, it follows that the mother-substance is represented by the formula $C_{10}H_{11}NO_3$. He describes a beautifully crystalline phenylcarbamic di-ester whose analysis leads sharply to this formula. Abel adduces strong evidence to show that adarenalin and suprarenin are modifications of epinephrin. All of these substances behave in the same manner toward solutions of silver nitrate and other oxidizing salts, all form iron and other metallic derivatives, all are capable of being acetylated and benzoated, all behave similarly and characteristically when heated under pressure, and all can be made to yield a peculiar basic substance of aconiine-piperidin odor and a black pigment of acid character. Analytic data are given to show that suprarenin is probably oxidized epinephrin $C_{10}H_{11}NO_4$, and the power of adarenalin promptly to reduce Fehling's solution is cited as distinguishing this substance from the native principle. Abel very properly objects to the adoption of the formula $C_9H_{13}NO_3$ for adarenalin, since the percentage of carbon required differs from the percentages found by analysis by 1.13 and 1.38; in fact, there is no rational formula that will meet Aldrich's analytic data. Takamine[4] does not hesitate after Aldrich's publication to make known his method of preparation. The finely divided capsules are extracted with water and the aqueous fluid heated for an hour at 90° C. for the coagulation of the proteids. The precipitate is filtered off, the filtrate is concentrated under diminished pressure, and the residue taken up in methyl alcohol. This alcoholic fluid is evaporated under diminished pressure and the adarenalin finally thrown out with ammonia. The analysis of this product leads Takamine to the formula $C_{10}H_{15}NO_3$. This formula differs from the one assigned by Aldrich to the group CH_2, yet Takamine states that Aldrich has proved the identity of the two preparations by analysis. Abel[1] shows that adarenalin can easily be converted into epinephrin by the action of acids. It is only necessary to dissolve the crystalline substance in strong hydrochloric acid, and to add alcohol and ether, when a substance is precipitated having all of the essential qualitative characters which have been ascribed to epinephrin hydrochlorate. Abel also notes that adarenalin contains phosphorus and continually gives off an alkaline gas when exposed to air. Moreover, he calls attention to a difference of 1.53 in carbon between the percentage found by Takamine and that required by the formula. It seems to us somewhat curious that four chemists have analyzed what is claimed to be a perfectly pure and stable crystalline body [adrenalin], yet no one has been able to assign a formula that meets the requirements of his analysis. All writers upon this subject agree that the native principle will not reduce Fehling's solution.[2] Abel[3] finds that extremely dilute solutions of adarenalin promptly reduce Fehling's solution, and offers this as evidence that adarenalin is not the native principle but probably a reduction product formed by the action of sulfureted hydrogen employed in Aldrich's process. However, Aldrich[4] claims that, contrary to the opinions of all the authorities cited, an aqueous extract of the gland will reduce Fehling's solution. He sums up the results of his work as follows: (1) The active principle obtained in the form of an extract without the use of an oxidizing or a reducing agent will reduce Fehling's solution about proportionately to the amount of adrenalin present; (2) the mother-liquor from adrenalin does not reduce Fehling's solution except in comparatively large amounts; (3) adrenalin prepared without the use of a reducing agent will reduce Fehling's solution; (4) adrenalin, the copper-sulfate-reducing body, and the blood-pressure-raising body are identical.

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[1] Am. Jour. of Physiol., 5, 457.

[2] Zeit. f. d. gesam. Biochem., 1, 243.

[3] Johns Hopkins Hosp. Bull., Nov., 1901.

[4] Am. Jour. of Pharm., 73, 523.

Hayashi[5] reports further observations on the **chemical nature of tetanus-toxin,** and concludes that it is neither a globulin nor an albumin, but is a primary albumose; thus it is but partly precipitated by magnesium sulfate, precipitation with alcohol does not alter its physical or toxic characters, it is precipitated by half saturation with ammonium sulfate, etc. The author also states that the tetanus bacilli do not grow in culture-mediums free from proteid.

Uric Acid.—Soetbur and Ibrahim[6] find that a solution of uric acid taken per os causes neither increase in the uric acid of the urine nor change in nitrogenous metabolism. There is under these conditions an increase in the total nitrogen of the feces, but this was shown not to be due to the presence of uric acid. It thus appears that uric acid when taken per os is not absorbed. When injected subcutaneously, the case is quite different. There is an immediate rise in the uric acid of the urine, about 80 % of that ingested appearing the first day. There is also a marked increase in the total nitrogen of the urine. Soetbur and Ibrahim conclude that circulating uric acid is principally excreted as such, not as urea; that it poisons the organism and causes an increased output both of total nitrogen and uric acid over that ingested.

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[1] Johns Hopkins Hosp. Bull., Feb.-Mar., 1902.

[2] Fraenkel, Wien. med. Blätt., 1896, No. 26, 409; Moore, Jour. of Physiol., 7. xiv; Metzger, Diss. Würtzburg, 1897; v. Fürth, Zeit. f. physiol. chem., 24, 142.

[3] Loc. cit.

[4] Am. Jour. of Physiol., 7, 359.

[5] Arch. f. exper. Path. u. Pharm., 47, p. 9.

[6] Zeit. f. physiol. Chem., 35, 1.

Wiener[1] contributes a long and important article on the **synthesis of uric acid in animal organism.** In his review of the present views on the subject of the formation of uric acid the author calls attention to the fact that it is generally supposed that this substance is formed by synthetic processes (from urea or ammonia and some nonnitrogenous body like lactic acid) in birds, while in mammals it is formed by the oxidation of purin bases. It has been shown, however, that uric acid is formed in birds to a slight extent by oxidation; Wiener shows in this paper that part of the uric acid in mammals is formed by synthetic processes. Hence the difference between the two classes of animals is one of degree rather than one of kind. One line of evidence for the synthetic formation of uric acid in mammals is obtained from experiments on "surviving organs." If the liver is finely minced and mixed with normal saline solution and allowed to remain for some time at 40° C., the amount of uric acid is increased; there is also a slight increase of uric acid when the spleen or thymus is similarly treated. If, however, the minced spleen or thymus is mixed with the liver, the increase in uric acid is very much greater. When an alcoholic extract of the spleen or thymus (such an extract contains neither nuclein nor xanthin bases) is added to the liver, a similar increase in uric acid is found. From the foregoing experiments the author draws the conclusion that the spleen and thymus contain certain substances (other than the purin bases) which may be synthetized by the liver-cells into uric acid; the spleen and thymus are able to oxidize the nucleins present to uric acid, but are not able to synthetize the other substances. The author next performed a series of experiments upon birds in order to determine what substances can be built up into uric acid. When 3 gm. of urea were given per os to a hen, nearly all of it appeared in the excrement as uric acid; when the same amount was given subcutaneously, the uric acid was relatively but slightly increased. In the latter case the body did not seem able to produce, in a short time, a sufficient amount of nonnitrogenous substance to unite with the urea. In the next experiments urea was given subcutaneously, and the effect of feeding various bodies studied. It was found that when hydracrylic, malonic, tartronic, or mesoxalic acid was fed, the amount of uric acid formed was very near the theoretic amount; after glycerin and lactic, pyruvic, glyceric, and β-oxybutyric acids the increase varied from 37 % to 45 % of the amount expected. There was no increase after propionic, succinic, malic, or butyric acids. Thus most of the substances with a chain of 3 C atoms caused an increased formation of uric acid, while most of those with 4 C atoms were inactive in this respect. An increase in the formation of uric acid was also caused, in similar experiments, by ordinary articles of diet—proteids, dextrose, and fats (olive oil). Finally, similar experiments were performed upon mammals (including man) and with the minced livers of mammals; it was found that some of the above substances (especially sodium malonate, glycerin, and dialuric and tartronic acids) caused an increased formation of uric acid; the increase was not as great as would theoretically be expected, but still the experiments show that the liver of mammals has the power of synthetizing uric acid from urea and certain nonnitrogenous substances.

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[1] Beitr. zur chem. Physiol. u. Path., 2, p. 2.

The influence of various acids upon **uric acid excretion** has been studied by Ulrici[1]; benzoic and gallic acids caused a decrease, while the closely related salicylic acid caused an increase in the amount of uric acid excreted. Quinic and tannic acids had no effect.

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[1] Arch. f. exper. Path. u. Pharm., 46, p. 321.

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